Resuscitation (2005) **67**, 271—291

RESUSCITATION

www.elsevier.com/locate/resuscitation

Part 6: Paediatric basic and advanced life support

International Liaison Committee on Resuscitation

The ILCOR Paediatric Task Force included expert reviewers from Africa, Asia, Australia, Asia, Europe, North America, and South America. These experts reviewed 45 topics related to paediatric resuscitation. Topics were selected from previous recommendations (the *ECC Guidelines 2000*),[1,2] emerging science, and newly identified issues. Some well-established topics without controversies or new evidence (e.g. adenosine for the treatment of supraventricular tachycardia (SVT)) are not included in this document.

Evidence-based worksheets on some topics were prepared and discussed but are not included here because there was insufficient evidence (e.g. fibrinolytics in cardiac arrest,[W13] securing the tracheal tube in children[W1], use of impedance threshold device in children,[W2] sodium bicarbonate for prolonged resuscitation attempts[W34]) or because no new evidence was found (e.g. evaluation of capillary refill,[W10] ventilation before naloxone,[W18] delayed volume resuscitation in trauma,[W17] use of hypertonic saline in shock[W16]).

The following is a summary of the most important changes in recommendations for paediatric resuscitation since the last ILCOR review in 2000.[1,2] The scientific evidence supporting these recommendations is summarised in this document:

- Emphasis on the quality of CPR is increased: ''Push hard, push fast, minimise interruptions; allow full chest recoil, and don't hyperventilate''.
  - Recommended chest compression—ventilation ratio:
    - o for one lay rescuer: 30:2;
    - o for healthcare providers performing two-rescuer CPR: 15:2.
  - Either the two- or one-hand technique is acceptable for chest compressions in children.
  - One initial shock followed by immediate CPR for attempted defibrillation instead of three stacked shocks.
- Biphasic attenuated shocks with an automated external defibrillator (AED) are acceptable for children >1 year.
- Routine use of high-dose adrenaline is no longer recommended.
- Either cuffed or uncuffed tracheal tubes are acceptable in infants and children.
- Exhaled CO_2 monitoring is recommended for confirmation of tracheal tube placement and during transport.
- Consider induced hypothermia for patients who remain comatose following resuscitation.
- Emphasis is increased on intravascular (intravenous (IV) and intraosseous (IO)) rather than tracheal administration of drugs.

The ILCOR Paediatric Task Force re-evaluated the definitions of newborn, infant, child, and adult. These definitions are somewhat arbitrary but are important because some recommendations for treatment differ according to patient size and the most likely aetiology of arrest. The distinction between child and adult victims has been deemphasised by the recommendation of a universal compression—ventilation ratio for lay rescuers and the same chest compression technique for lay rescuers of children and adults. Some differences in treatment recommendations remain between the

doi:10.1016/j.resuscitation.2005.09.020

newborn and infant and between an infant and child, but those differences are chiefly linked to resuscitation training and practice. They are noted below.

Identified knowledge gaps in paediatric resuscitation include:

- Sensitive and specific indicators of cardiac arrest that lay rescuers and healthcare providers can recognise reliably.
- Effectiveness of aetiology-based versus age-based resuscitation sequences.
- The ideal ratio of chest compressions to ventilations during CPR.
- Mechanisms to monitor and optimise quality of CPR during attempted resuscitation.
- Best methods for securing a tracheal tube.
- Clinical data on the safety and efficacy of automated external defibrillators (AEDs).
- Clinical data on the safety and efficacy of the laryngeal mask airway (LMA) during cardiac arrest.
- The benefits and risks of supplementary oxygen during and after CPR.
- Clinical data on antiarrhythmic and pressor medications during cardiac arrest.
- Data on induced hypothermia in paediatric cardiac arrest.
- The identification and treatment of post-arrest myocardial dysfunction.
- The use of fibrinolytics and anticoagulants in cardiac arrest.
- Use of emerging technologies for assessment of tissue perfusion.
- Predictors of outcome from cardiac arrest.

Initial steps of CPR

The *ECC Guidelines 2000*[1] recommended that lone rescuers of *adult* victims of cardiac arrest phone the emergency medical services (EMS) system and get an AED (''call first'') before starting CPR. The lone rescuer of an unresponsive *infant or child* victim was instructed to provide a brief period of CPR before leaving the victim to phone for professional help and an AED (''call fast''). These sequence differences were based on the supposition that cardiac arrest in adults is due primarily to ventricular fibrillation (VF) and that a hypoxic-ischaemic mechanism is more common in children. But this simplistic approach may be inaccurate and may not provide the ideal rescue sequence for many victims of cardiac arrest. Hypoxic-ischaemic arrest may occur in adults, and VF may be the cause of cardiac arrest in up to 7% to 15% of infants and children. Resuscitation results might be improved if the sequence of lay rescuer CPR actions, (i.e. the priority of phoning for professional help, getting an AED, and providing CPR) is based on the aetiology of cardiac arrest rather than age.

The pulse check was previously eliminated as an assessment for the lay rescuer. There is now evidence that healthcare professionals may take too long to check for a pulse and may not determine the presence or absence of the pulse accurately. This may lead to interruptions in chest compressions and affect the quality of CPR.

Experts reviewed the data on the technique of rescue breathing for infants and the two-thumb-encircling hands versus two-finger chest compression techniques for infants.

One of the most challenging topics debated during the 2005 Consensus Conference was the compression—ventilation ratio. The scientific evidence on which to base recommendations was sparse, and it was difficult to arrive at consensus. Evidence was presented that the ratio should be higher than 5:1, but the optimal ratio was not identified. The only data addressing a compression—ventilation ratio greater than 15:2 came from mathematical models. The experts acknowledged the educational benefit of simplifying training for lay rescuers (specifically one-rescuer CPR) by adopting a single ratio for infants, children, and adults with the hope that simplification might increase the number of bystanders who will learn, remember, and perform CPR. On this basis experts agreed that this single compression—ventilation ratio should be 30:2. Healthcare providers typically will be experienced in CPR and practice it frequently. This group of experienced providers will learn two-person CPR, and for them the recommended compression—ventilation ratio for two rescuers is 15:2.

Some laypeople are reluctant to perform mouth-to-mouth ventilation. For treatment of cardiac arrest in infants and children, chest compressions alone are better than no CPR but not as good as a combination of ventilations and compressions.

In the past one-handed chest compressions were recommended for CPR in children. A review of the evidential basis for this recommendation was conducted. From an educational standpoint, we agree that it will simplify training to recommend a single technique for chest compressions for children and adults.

Activating emergency medical services and getting the AED
W4

Consensus on science. Most cardiac arrests in children are caused by asphyxia (LOE 4).[3–6]

Observational studies of non-VF arrests in children show an association between bystander CPR and intact neurological outcome (LOE 4).[6–8] Animal studies show that in asphyxial arrest, chest compressions plus ventilation CPR is superior to either chest compression-only CPR or ventilation-only CPR (LOE 6).[9]

Observational studies of children with VF report good (17% to 20%) rates of survival after early defibrillation (LOE 4).[5,6,10] The merits of ''call first'' versus ''call fast'' CPR sequences have not been studied adequately in adults or children with cardiac arrest of asphyxial or VF etiologies. Three animal studies (LOE 6)[9,11,12] show that even in prolonged VF, CPR increases the likelihood of successful defibrillation, and seven adult human studies (LOE 7)[13–19] document improved survival with the combination of CPR with minimal interruptions in chest compression and early defibrillation.

Treatment recommendation. A period of immediate CPR before phoning emergency medical services (EMS) and getting the AED (''call fast'') is indicated for most paediatric arrests because they are presumed to be asphyxial or prolonged. In a witnessed sudden collapse (e.g. during an athletic event), the cause is more likely to be VF, and the lone rescuer should phone for professional help and get the AED (when available) before starting CPR. Rescuers should perform CPR with minimal interruptions in chest compressions until attempted defibrillation.

In summary, the priorities for *unwitnessed* or non-sudden collapse in children are as follows:

- Start CPR immediately.
- Activate EMS/get the AED.

The priorities for *witnessed* sudden collapse in children are as follows:

- Activate EMS/get the AED.
- Start CPR.
- Attempt defibrillation.

Pulse check
W5A,W5B

Consensus on science. Ten studies (LOE 2[20,21]; LOE 4[22–26]; LOE 5[27]; LOE 6[28,29]) show that lay rescuers[23,25,30] and healthcare providers[20,21,24,26–29] show that rescuers are often unable to accurately determine the presence of a pulse within 10 s. Two studies in infants (LOE 5)[31,32] reported that rescuers detected cardiac activity rapidly by direct chest auscultation but were biased because they knew that the infants were healthy.

Treatment recommendation. Lay rescuers should start chest compressions for an unresponsive infant or child who is not moving or breathing. Healthcare professionals may also check for a pulse but should proceed with CPR if they cannot feel a pulse within 10 s or are uncertain if a pulse is present.

Ventilations in infants
W7A,W7B

Consensus on science. One LOE 5[33] study and 10 LOE 7[34–43] reports assessed a mouth-to-nose ventilation technique for infants. The LOE 5 study[33] is an anecdotal report of three infants ventilated with mouth-to-nose technique. The LOE 7 reports describe postmortem anatomy,[34] physiology of nasal breathing,[35–37] related breathing issues,[38,39] and measurements of infants' faces compared with the measurement of adult mouths.[40–43] There is great variation in these measurements, probably because of imprecise or inconsistent definitions.

Treatment recommendation. There are no data to justify a change from the recommendation that the rescuer attempt mouth-to—mouth-and-nose ventilation for infants. Rescuers who have difficulty achieving a tight seal over the mouth and nose of an infant, however, may attempt either mouth-to-mouth or mouth-to-nose ventilation (LOE 5).[33]

Circumferential versus two-finger chest compressions
W9A,W9B

Consensus on science. Two manikin (LOE 6)[44,45] and two animal (LOE 6)[46,47] studies showed that the two thumb-encircling hands technique of chest compressions with circumferential thoracic squeeze produces higher coronary perfusion pressures and more consistently correct depth and force of compression than the two-finger technique.

Case reports (LOE 5)[48,49] of haemodynamic monitoring in infants receiving chest compressions showed higher systolic and diastolic arterial pressures in the two-thumb encircling hands technique compared with the two-finger technique.

Treatment recommendation. The two thumb-encircling hands chest compression technique with thoracic squeeze is the preferred technique for two-rescuer infant CPR. The two-finger technique is recommended for one-rescuer infant CPR to facilitate rapid transition between compression and ventilation to minimise interruptions in chest compressions. It remains an acceptable alternative method of chest compressions for two rescuers.

One- versus two-hand chest compression technique
W276

Consensus on science. There are no outcome studies that compare one- versus two-hand compressions of the chest in children. One (LOE 6)[50] study reported higher pressures generated in child manikins using the two-hand technique to compress over the lower part of the sternum to a depth of approximately one-third the anterior-posterior diameter of the chest. Rescuers reported that this technique was easy to perform.

Treatment recommendation. Both the one- and two-hand techniques for chest compressions in children are acceptable provided that rescuers compress over the lower part of the sternum to a depth of approximately one-third the anterior-posterior diameter of the chest. To simplify education, rescuers can be taught the same technique (i.e. two-hand) for adult and child compressions.

Compression—ventilation ratio
W3A,W3B,W3C

Consensus on science. There are insufficient data to identify an optimal compression—ventilation ratio for CPR in children. Manikin studies (LOE 6)[51–54] have examined the feasibility of compression—ventilation ratios of 15:2 and 5:1. Lone rescuers cannot deliver the desired number of chest compressions per minute at a ratio of 5:1. A mathematical model (LOE 7)[55] supports compression—ventilation ratios higher than 5:1 for infants and children.

Two animal (LOE 6)[56,57] studies show that coronary perfusion pressure, a major determinant of success in resuscitation, declines with interruptions in chest compressions. In addition, once compressions are interrupted, several chest compressions are needed to restore coronary perfusion pressure. Frequent interruptions of chest compressions (e.g. with a 5:1 compression—ventilation ratio) prolongs the duration of low coronary perfusion pressure. Long interruptions in chest compressions have been documented in manikin studies (LOE 6)[58,59] and both in- and out-of-hospital adult CPR studies (LOE 7).[60,61] These interruptions reduce the likelihood of a return of spontaneous circulation (LOE 7).[62–64]

Five animal (LOE 6)[9,56,57,65,66] studies and one review (LOE 7)[67] review suggest that ventilations are relatively less important in victims with VF or pulseless ventricular tachycardia (VT) cardiac arrest than in victims with asphyxia-induced arrest. But even in asphyxial arrest, few ventilations are needed to maintain an adequate ventilation-perfusion ratio in the presence of the low cardiac output (and, consequently low pulmonary blood flow) produced by chest compressions.

Treatment recommendation. For ease of teaching and retention, a universal compression—ventilation ratio of 30:2 is recommended for the lone rescuer responding to infants (for neonates see Part 7: ''Neonatal Resuscitation''), children, and adults. For healthcare providers performing two-rescuer CPR, a compression—ventilation ratio of 15:2 is recommended. When an advanced airway is established (e.g. a tracheal tube, Combitube, or laryngeal mask airway (LMA)), ventilations are given without interrupting chest compressions.

Some CPR versus no CPR
W8

Consensus on science. Numerous reports (LOE 5)[4,5,8,68–70] document survival of children after cardiac arrest when bystander CPR was provided. Bystander CPR in these reports included rescue breathing alone, chest compressions alone, or a combination of compressions and ventilations.

One prospective and three retrospective studies of adult VF (LOE 7)[71–74] and numerous animal studies of VF cardiac arrest (LOE 6)[56,57,66,75–79] document comparable long-term survival after chest compressions alone or chest compressions plus ventilations, and both techniques result in better outcomes compared with no CPR. In animals with asphyxial arrest (LOE 6),[9] the more common mechanism of cardiac arrest in infants and children, best results are achieved with a combination of chest compressions and ventilations. But resuscitation with either ventilations only or chest compressions only is better than no CPR.

Treatment recommendation. Bystander CPR is important for survival from cardiac arrest. Trained rescuers should be encouraged to provide both ventilations and chest compressions. If rescuers are reluctant to provide rescue breaths, however, they should be encouraged to perform chest compressions alone without interruption.

Disturbances in cardiac rhythm

Evidence evaluation for the treatment of haemodynamically stable arrhythmias focused on vagal manoeuvres, amiodarone, and procainamide. There were no new data to suggest a change in the indications for vagal manoeuvres or procainamide. Several case series described the safe and effective use of amiodarone in children, but these studies involved selected patient populations (often with postoperative arrhythmias) treated by experienced providers in controlled settings. Although there is no change in the recommendation for amiodarone as a treatment option in children with stable arrhythmias, providers are encouraged to consult with an expert knowledgeable in paediatric arrhythmias before initiating drug therapy.

There is insufficient evidence to identify an optimal shock waveform, energy dose, and shock strategy (e.g. fixed versus escalating shocks, one versus three stacked shocks) for defibrillation. The new recommendation for the sequence of defibrillation in children is based on extrapolated data from adult and animal studies with biphasic devices, data documenting the high rates of success for first shock conversion of VF with biphasic waveforms, and knowledge that interruption of chest compressions reduces coronary perfusion pressure. Thus, a one-shock strategy may be preferable to the three-shock sequence recommended in the *ECC Guidelines 2000*.[2] For further details, see Part 3: Defibrillation.

Many, but not all, AED algorithms have been shown to be sensitive and specific for recognising shockable arrhythmias in children. A standard AED (adult AED with adult pad-cable system) can be used for children older than about 8 years and weighing more than about 25 kg. Many manufacturers now provide a method for attenuating the energy delivered to make the AED suitable for smaller children (e.g. use of a pad-cable system or an AED with a key or switch to select a smaller dose).

Management of supraventricular tachycardias

If the child with SVT is haemodynamically stable, we recommend early consultation with a paediatric cardiologist or other physician with appropriate expertise. This recommendation is common for all of the SVT topics below.

Vagal manoeuvres for SVT
W36

Consensus on science. One prospective (LOE 3)[80] and nine observational studies (LOE 4[81]; LOE 5[82,83]; LOE 7[84–89]) show that vagal manoeuvres are effective in terminating SVT in children. There are reports of complications from carotid sinus massage and application of ice to the face to stimulate the diving reflex (LOE 5),[90,91] but virtually none from the Valsalva manoeuvre.

Treatment recommendation. The Valsalva manoeuvre and ice application to the face may be used to treat haemodynamically stable SVT in infants and children. When performed correctly, these manoeuvres can be initiated quickly and safely and without altering subsequent therapies if they fail.

Amiodarone for haemodynamically stable SVT
W38

Consensus on science. One prospective (LOE 3)[92] and 10 observational (LOE 5)[93–102] studies show that amiodarone is effective for treating SVT in children. A limitation of this evidence is that most of the studies in children describe treatment for post-operative junctional ectopic tachycardia.

Treatment recommendation. Amiodarone may be considered in the treatment of haemodynamically stable SVT refractory to vagal manoeuvres and adenosine. Rare but significant acute side effects include bradycardia, hypotension, and polymorphic VT (LOE 5).[103–105]

Procainamide for haemodynamically stable SVT
W37

Consensus on science. Experience with procainamide in children is limited. Twelve LOE 5[106–117] and four LOE 6[118–121] observational studies show that procainamide can terminate SVT that is resistant to other drugs. Most of these reports include mixed populations of adults and children. Hypotension following procainamide infusion results from its vasodilator action rather than a negative inotropic effect (LOE 5[122,123]; LOE 6[124]).

Treatment recommendation. Procainamide may be considered in the treatment of haemodynamically stable SVT refractory to vagal manoeuvres and adenosine.

Management of stable wide-QRS tachycardia

If a child with wide-QRS tachycardia is haemodynamically stable, early consultation with a paediatric cardiologist or other physician with appropriate expertise is recommended. In general, amiodarone and procainamide should not be administered together because their combination may increase risk of hypotension and ventricular arrhythmias.

Amiodarone
W39A,W39B,W40

Consensus on science. One case series (LOE 5)[125] suggests that wide-QRS tachycardia in children is more likely to be supraventricular than ventricular in origin. Two prospective studies (LOE 3)[92,126] and 13 case series (LOE 5)[93–102,127–129] show that amiodarone is effective for a wide variety of tachyarrhythmias in children. None of these reports specifically evaluates the role of amiodarone in the setting of a stable, unknown wide-complex tachycardia.

Treatment recommendation. Wide-QRS tachycardia in children who are stable may be treated as SVT. If the diagnosis of VT is confirmed, amiodarone should be considered.

Procainamide for stable VT
W35

Consensus on science. Twenty (LOE 5)[106,115,123,130–146] and two LOE 6[118,124] observational studies, primarily in adults, but including some children show that procainamide is effective in the treatment of stable VT.

Treatment recommendation. Procainamide may be considered in the treatment of haemodynamically stable VT.

Management of unstable VT

Amiodarone
W39A,W40

Consensus on science. In small paediatric case series (LOE 3[100]; LOE 5[93,95,97,99,147–149]) and extrapolation from animal (LOE 6)[150,151] and adult (LOE 7)[152–165] studies, amiodarone is safe and effective for haemodynamically unstable VT in children.

Treatment recommendation. Synchronised cardioversion remains the treatment of choice for unstable VT. Amiodarone may be considered for treatment of haemodynamically unstable VT.

Paediatric defibrillation

For additional information about consensus on science and treatment recommendations for defibrillation (e.g. one versus three stacked shock sequences and sequence of CPR first versus defibrillation first), see Part 3: ''Defibrillation.''

Manual and automated external defibrillation
W41A,W41B

Consensus on science. The ideal energy dose for safe and effective defibrillation in children is unknown. Extrapolation from adult data (LOE 1[166,167]; LOE 2[168–170]) and paediatric animal studies (LOE 6)[171–173] shows that biphasic shocks are at least as effective as monophasic shocks and produce less postshock myocardial dysfunction. One LOE 5[174] and one LOE 6[171] study show that an initial monophasic or biphasic shock dose of 2 J kg^{-1} generally terminates paediatric VF. Two paediatric case series (LOE 5)[175,176] report that doses >4 J kg^{-1} (up to 9 J kg^{-1}) have effectively defibrillated children <12 years, with negligible adverse effects.

In five animal studies (LOE 6)[172,173,177–179] large (per kilogram) energy doses caused less myocardial damage in young hearts than in adult hearts. In three animal studies (LOE 6)[173,179,180] and one small paediatric case series (LOE 5),[176] a 50-J biphasic dose delivered through a paediatric pad/cable system terminated VF and resulted in survival. One piglet (13–26 kg) study (LOE 6)[179] showed that paediatric biphasic AED shocks (50/75/86 J) terminated VF and caused less myocardial injury and better outcome than adult AED biphasic shocks (200/300/360 J).

Treatment recommendation. The treatment of choice for paediatric VF/pulseless VT is prompt defibrillation, although the optimum dose is unknown. For manual defibrillation, we recommend an initial dose of 2 J kg^{-1} (biphasic or monophasic waveform). If this dose does not terminate VF, subsequent doses should be 4 J kg^{-1}.

For automated defibrillation, we recommend an initial paediatric attenuated dose for children 1–8 years and up to about 25 kg and 127 cm in length. There is insufficient information to recommend for or against the use of an AED in infants <1 year. A variable dose manual defibrillator or an AED able to recognise paediatric shockable rhythms and equipped with dose attenuation are preferred; if such a defibrillator is not available, a standard AED with standard electrode pads may be used. A standard AED (without a dose attenuator) should be used for children ≥25 kg (about 8 years) and adolescent and adult victims.

Management of shock-resistant VF/pulseless VT

Amiodarone
W20,W21A,W21B

Consensus on science. Evidence extrapolated from three (LOE 1) studies in adults (LOE 7 when applied to children)[154,159,181] shows increased survival to hospital admission but not discharge when amiodarone is compared with placebo or lidocaine for shock-resistant VF. One study in children (LOE

3)[100] showed effectiveness of amiodarone for life-threatening ventricular arrhythmias.

Treatment recommendation. Intravenous amiodarone can be considered as part of the treatment of shock-refractory or recurrent VT/VF.

Airway and ventilation

Maintaining a patent airway and ventilation are fundamental to resuscitation. Adult and animal studies during CPR suggest detrimental effects of hyperventilation and interruption of chest compressions. For children requiring airway control or ventilation for short periods in the out-of-hospital setting, bag-valve-mask (BVM) ventilation produces equivalent survival rates compared with ventilation with tracheal intubation.

The risks of tracheal tube misplacement, displacement, and obstruction are well recognized, and an evidence-based review led to a recommendation that proper tube placement and patency be monitored by exhaled CO_2 throughout transport. A review also found that cuffed tracheal tubes could be used safely even in infants.

Following the return of spontaneous circulation from cardiac arrest, toxic oxygen by-products (reactive oxygen species, free radicals) are produced that may damage cell membranes, proteins, and DNA (reperfusion injury). There are no clinical studies in children outside the newborn period comparing different concentrations of inspired oxygen during and immediately after resuscitation, and it is difficult to differentiate sufficient from excessive oxygen therapy.

Bag-valve-mask ventilation
W6

Consensus on science. One out-of-hospital paediatric randomised controlled study (LOE 1)[182] in an EMS system with short transport times showed that BVM ventilation compared with tracheal intubation resulted in equivalent survival to hospital discharge rates and neurological outcome in children requiring airway control, including children with cardiac arrest and trauma.

One study in paediatric cardiac arrest (LOE 4)[183] and four studies in children with trauma (LOE 3[184,185]; LOE 4[186,187]) found no advantage of tracheal intubation over BVM ventilation.

Treatment recommendation. In the out-of-hospital setting with short transport times, BVM ventilation is the method of choice for children who require ventilatory support. When transport times are long, the relative benefit versus potential harm of tracheal intubation compared with BVM ventilation is uncertain. It is affected by the level of training and experience of the healthcare professional and the availability of exhaled CO_2 monitoring during intubation and transport.

Advanced airways

Advanced airways include the tracheal tube, the Combitube, and the LMA. Experts at the 2005 Consensus Conference reviewed the available evidence on use of the tracheal tube and LMA in infants and children. There were no data on use of the Combitube in this age group.

Cuffed versus uncuffed tracheal tubes
W11A,W11B

Consensus on science. One randomised controlled trial (LOE 2),[188] three prospective cohort studies (LOE 3),[189–191] and one cohort study (LOE 4)[192] document no greater risk of complications for children <8 years when using cuffed tracheal tubes compared with uncuffed tubes in the operating room and intensive care unit.

Evidence from one randomised controlled trial (LOE 2)[188] and one small, prospective controlled study (LOE 3)[193] showed some advantage in cuffed over uncuffed tracheal tubes in children in the paediatric anaesthesia and intensive care settings, respectively.

Treatment recommendation. Cuffed tracheal tubes are as safe as uncuffed tubes for infants (except newborns) and children if rescuers use the correct tube size and cuff inflation pressure and verify tube position. Under certain circumstances (e.g. poor lung compliance, high airway resistance, and large glottic air leak), cuffed tracheal tubes may be preferable.

Laryngeal mask airway
W26A,W26B

Consensus on science. There are no studies examining the use of the LMA in children during cardiac arrest. Evidence extrapolated from paediatric anaesthesia shows a higher rate of complications with LMAs in smaller children compared with LMA experience in adults. The complication rate decreases with increasing operator experience (LOE 7).[194,195] Case reports document that the LMA can be helpful for management of the difficult airway.

Treatment recommendation. There are insufficient data to support or refute a recommendation for the routine use of an LMA for children in cardiac arrest. The LMA may be an acceptable initial alternative airway adjunct for experienced providers during paediatric cardiac arrest when tracheal intubation is difficult to achieve.

Confirmation of tube placement

Exhaled CO_2
W25

Consensus on science. Misplaced, displaced, or obstructed tracheal tubes are associated with a high risk of death. No single method of tracheal tube confirmation is always accurate and reliable. One study (LOE 3)[196] showed that clinical assessment of tracheal tube position (observation of chest wall rise, mist in the tube, and auscultation of the chest) can be unreliable for distinguishing oesophageal from tracheal intubation.

In three studies (LOE 5),[197–199] when a perfusing cardiac rhythm was present in infants >2 kg and children, detection of exhaled CO_2 using a colourmetric detector or capnometer had a high sensitivity and specificity for tracheal tube placement. In one study (LOE 5)[198] during cardiac arrest, the sensitivity of exhaled CO_2 detection for tracheal tube placement was 85% and specificity 100%. Both with a perfusing rhythm and during cardiac arrest, the presence of exhaled CO_2 reliably indicates tracheal tube placement, but the absence of exhaled CO_2 during cardiac arrest does not prove tracheal tube misplacement.

Treatment recommendation. In all settings (i.e. prehospital, emergency departments, intensive care units, operating rooms), confirmation of tracheal tube placement should be achieved using detection of exhaled CO_2 in intubated infants and children with a perfusing cardiac rhythm. This may be accomplished using a colourmetric detector or capnometry. During cardiac arrest, if exhaled CO_2 is not detected, tube position should be confirmed using direct laryngoscopy.

Oesophageal detector device
W23

Consensus on science. A study in the operating room (LOE 2)[200] showed that the oesophageal detector device (EDD) was highly sensitive and specific for correct tracheal tube placement in children weighing >20 kg with a perfusing cardiac rhythm. There have been no studies of the EDD in children during cardiac arrest. A paediatric animal study (LOE 6)[201] showed only fair results with the EDD, but accuracy improved with use of a larger syringe. The same animal study showed no difference when the tracheal tube cuff was either inflated or deflated.

Treatment recommendation. The EDD may be considered for confirmation of tracheal tube placement in children weighing >20 kg.

Confirmation of tracheal tube placement during transport
W24

Consensus on science. Studies (LOE 1[202]; LOE 7[203]) have documented the high rate of inadvertent displacement of tracheal tubes during prehospital transport. There are no studies to evaluate the frequency of these events during intra- or inter-hospital transport.

Two studies (LOE 5)[204,205] show that in the presence of a perfusing rhythm, exhaled CO_2 detection or measurement can confirm tracheal tube position accurately during transport. In two animal studies (LOE 6),[206,207] loss of exhaled CO_2 detection indicated tracheal tube displacement more rapidly than pulse oximetry. On the basis of one case series (LOE 5),[204] continuous use of colourmetric exhaled CO_2 detectors may not be reliable for long (>30 min) transport duration.

Treatment recommendation. We recommend monitoring tracheal tube placement and patency in infants and children with a perfusing rhythm by continuous measurement or frequent intermittent detection of exhaled CO_2 during prehospital and intra- and inter-hospital transport.

Oxygen

Oxygen during resuscitation
W14A,W14B

Consensus on science. Meta-analyses of four human studies (LOE 1)[208–211] showed a reduction in mortality rates and no evidence of harm in newborns resuscitated with air compared with 100% oxygen (see Part 7: Neonatal Resuscitation). The two largest studies,[210,211] however, were not blinded, so results should be interpreted with caution. Two animal studies (LOE 6)[212,213] suggest that ventilation with room air may be superior to 100% oxygen during resuscitation from cardiac arrest, whereas one animal study (LOE 6)[214] showed no difference.

Treatment recommendation. There is insufficient information to recommend for or against the use of any specific inspired oxygen concentration during and immediately after resuscitation from cardiac arrest. Until additional evidence is published, we support healthcare providers' use of 100% oxygen during resuscitation (when available). Once circulation is restored, providers should monitor oxygen saturation and reduce the inspired oxygen concentration while ensuring adequate oxygen delivery.

Vascular access and drugs for cardiac arrest

Vascular access can be difficult to establish during resuscitation of children. Review of the evidence showed increasing experience with IO access and resulted in a de-emphasis of the tracheal route for drug delivery. Evidence evaluation of resuscitation drugs was limited by a lack of reported experience in children. There was little experience with vasopressin in children in cardiac arrest and inconsistent results in adult patients. In contrast, there was a good study in children showing no benefit and possibly some harm in using high-dose adrenaline for cardiac arrest.

Routes of drug delivery

Intraosseous access
W29

Consensus on science. Two prospective randomised trials in adults and children (LOE 3)[215,216] and six other studies (LOE 4[217]; LOE 5[218–220]; LOE 7[221,222]) document that IO access is safe and effective for fluid resuscitation, drug delivery, and blood sampling for laboratory evaluation.

Treatment recommendation. We recommend establishing IO access if vascular access is not achieved rapidly in any infant or child for whom IV drugs or fluids are urgently required.

Drugs given via tracheal tube
W32

Consensus on science. One study in children (LOE 2),[223] five studies in adults (LOE 2[224–226]; LOE 3[227,228]), and multiple animal studies (LOE 6)[229–231] indicate that atropine, adrenaline, naloxone, lidocaine, and vasopressin are absorbed via the trachea. Administration of resuscitation drugs into the trachea results in lower blood concentrations than the same dose given intravascularly. Furthermore, animal studies (LOE 6)[232–235] suggest that the lower adrenaline concentrations achieved when the drug is delivered by tracheal route may produce transient β-adrenergic effects. These effects can be detrimental, causing hypotension, lower coronary artery perfusion pressure and flow, and reduced potential for return of spontaneous circulation.

Treatment recommendation. Intravascular, including IO, injection of drugs is preferable to administration by the tracheal route. The recommended tracheal dose of atropine, adrenaline, or lidocaine is higher than the vascular dose and is as follows:

- Adrenaline $0.1\,\mathrm{mg\,kg^{-1}}$ (multiple LOE 6 studies).
- Lidocaine $2–3\,\mathrm{mg\,kg^{-1}}$ (LOE 3)[228] and multiple LOE 6 studies.
- Atropine $0.03\,\mathrm{mg\,kg^{-1}}$ (LOE 2)[224].

The optimal tracheal doses of naloxone or vasopressin have not been determined.

Drugs in cardiac arrest

Dose of adrenaline for cardiac arrest
W31A,W31B

Consensus on science. In four paediatric studies (LOE 2[236,237]; LOE 4[238,239]) there was no improvement in survival rates and a trend toward worse neurological outcome after administration of high-dose adrenaline for cardiac arrest. A randomised, controlled trial (LOE 2)[236] comparing high-dose with standard-dose adrenaline for the second and subsequent (''rescue'') doses in paediatric in-hospital cardiac arrest showed reduced 24-h survival rates in the high-dose adrenaline group. In subgroup analysis, survival rates in asphyxia and sepsis were significantly worse with high-dose rescue adrenaline.

Treatment recommendation. Children in cardiac arrest should be given $10\,\mu\mathrm{g\,kg^{-1}}$ of adrenaline as the first and subsequent intravascular doses. Routine use of high-dose ($100\,\mu\mathrm{g\,kg^{-1}}$) intravascular adrenaline is not recommended and may be harmful, particularly in asphyxia. High-dose adrenaline may be considered in exceptional circumstances (e.g. β-blocker overdose).

Vasopressin in cardiac arrest
W19A,W19B

Consensus on science. Based on a small series of children (LOE 5),[240] vasopressin given after adrenaline may be associated with return of spontaneous circulation after prolonged cardiac arrest. Animal data (LOE 6)[241,242] indicate that a combination of adrenaline and vasopressin may be beneficial. Adult data are inconsistent. Giving vasopressin after adult cardiac arrest (LOE 7)[243–247] has produced improved short-term outcomes (e.g. return of spontaneous circulation or survival to hospital admission) but no improvement in neurologically intact survival to hospital discharge when compared with adrenaline.

Treatment recommendation. There is insufficient evidence to recommend for or against the routine use of vasopressin during cardiac arrest in children.

Magnesium in cardiac arrest
W15

Consensus on science. The relationship between serum magnesium concentrations and outcome of CPR was analyzed in three studies in adults (LOE 3[248]; LOE 4[249]) and one animal study (LOE 6).[250] The first two studies indicated that a normal serum concentration of magnesium was associated with a higher rate of successful resuscitation, but it is unclear whether the association is causative. Six adult clinical studies (LOE 1[251]; LOE 2[252–255]; LOE 3[256]) and one study in an adult animal model (LOE 6)[257] indicated no significant difference in any survival end point in patients who received magnesium before, during, or after CPR.

Treatment recommendation. Magnesium should be given for hypomagnesaemia and torsades de pointes VT, but there is insufficient evidence to recommend for or against its routine use in cardiac arrest.

Postresuscitation care

Postresuscitation care is critical to a favourable outcome. An evidence-based literature review was performed on the topics of brain preservation and myocardial function after resuscitation from cardiac arrest. It showed the potential benefits of induced hypothermia on brain preservation, the importance of preventing or aggressively treating hyperthermia, the importance of glucose control, and the role of vasoactive drugs in supporting haemodynamic function.

Ventilation

Hyperventilation
W27

Consensus on science. One study in cardiac arrest patients (LOE 3)[258] and extrapolation from 12 other studies (LOE 6[259]; LOE 2[260]; LOE 3[261–267]; LOE 4[268]; LOE 5[269,270]) suggest that hyperventilation may cause decreased venous return to the heart and cerebral ischaemia and may be harmful in the comatose patient after cardiac arrest.

Treatment recommendation. Hyperventilation after cardiac arrest may be harmful and should be avoided. The target of postresuscitation ventilation is normocapnoea. Short periods of hyperventilation may be performed as a temporising measure for the child with signs of impending cerebral herniation.

Temperature control

Therapeutic hypothermia
W22B,W22C

Consensus on science. Immediately after resuscitation from cardiac arrest, children often develop hypothermia followed by delayed hyperthermia (LOE 5).[271] Hypothermia (32 °C—34 °C) may be beneficial to the injured brain. Although there are no paediatric studies of induced hypothermia after cardiac arrest, support for this treatment is extrapolated from:

- Two prospective randomised studies of adults with VF arrest (LOE 1[272]; LOE 2[273]).
- One study of newborns with birth asphyxia (LOE 2)[274].
- Numerous animal studies (LOE 6) of both asphyxial and VF arrest.
- Acceptable safety profiles in adults (LOE 7)[272,273] and neonates (LOE 7)[275–278] treated with hypothermia (32 °C—34 °C) for up to 72 h.

Treatment recommendation. Induction of hypothermia (32 °C—34 °C) for 12—24 h should be considered in children who remain comatose after resuscitation from cardiac arrest.

Treatment of hyperthermia
W22A,W22D

Consensus on science. Two studies (LOE 5)[271,279] show that fever is common after resuscitation from cardiac arrest, and three studies (LOE 7)[280–282] show that it is associated with worse outcome. Animal studies suggest that fever causes a worse outcome. One study (LOE 6)[283] shows that rats resuscitated from asphyxial cardiac arrest have a worse outcome if hyperthermia is induced within the first 24 h of recovery. In rats with global ischaemic brain injury (which produces endogenous fever), prevention of fever with a nonsteroidal anti-inflammatory drug (NSAID) class of antipyretic attenuated neuronal damage (LOE 6).[284,285]

Treatment recommendation. Healthcare providers should prevent hyperthermia and treat it aggressively in infants and children resuscitated from cardiac arrest.

Haemodynamic support

Vasoactive drugs
W33A,W33B,W33C,W33D

Consensus on science. Two studies in children (LOE 5),[286,287] multiple studies in adults (LOE 7[288—290]), and animal studies (LOE 6)[291—293] indicate that myocardial dysfunction is common after resuscitation from cardiac arrest. Multiple animal studies (LOE 6)[294—296] document consistent improvement in haemodynamics when selected vasoactive drugs are given in the post-cardiac arrest period. Evidence extrapolated from multiple adult and paediatric studies (LOE 7)[297—302] of cardiovascular surgical patients with low cardiac output documents consistent improvement in haemodynamics when vasoactive drugs are titrated in the period after cardiopulmonary bypass.

Treatment recommendation. Vasoactive drugs should be considered to improve haemodynamic status in the post-cardiac arrest phase. The choice, timing, and dose of specific vasoactive drugs must be individualised and guided by available monitoring data.

Blood glucose control

Treatment of hypoglycaemia and hyperglycaemia
W30A,W30B,W30C

Consensus on science. Adults with out-of-hospital cardiac arrest and elevated blood glucose on admission have poor neurological and survival outcomes (LOE 7).[303—308] In critically ill children, hypoglycaemia (LOE 5)[309] and hyperglycaemia (LOE 5)[310] are associated with poor outcome. It is unknown if the association of hyperglycaemia with poor outcome after cardiac arrest is causative or an epiphenomenon related to the stress response.

In critically ill adult surgical patients, (LOE 7)[311] strict glucose control improves outcome, but there are currently insufficient data in children showing that the benefit of tight glucose control outweighs the risk of inadvertent hypoglycaemia.

Several adult and animal studies (LOE 6)[312—316] and an adult clinical study (LOE 4)[317] show poor outcome when glucose is given immediately before or during cardiac arrest. It is unknown if there is harm in giving glucose-containing maintenance fluids to children after cardiac arrest.

Hypoglycaemia is an important consideration in paediatric resuscitation because:

- Critically ill children are hypermetabolic compared with baseline and have increased glucose requirement (6—8 $\mathrm{mg\,kg^{-1}\,min^{-1}}$) to prevent catabolism.
- The combined effects of hypoglycaemia and hypoxia/ischaemia on the immature brain (neonatal animals) appears more deleterious than the effect of either insult alone.[318]
- Four retrospective studies of human neonatal asphyxia show an association between hypoglycaemia and subsequent brain injury (LOE 4[319,320]; LOE 5[321,322]).

Treatment recommendation. Healthcare providers should check glucose concentration during cardiac arrest and monitor it closely afterward with the goal of maintaining normoglycaemia. Glucose-containing fluids are not indicated during CPR unless hypoglycaemia is present (LOE 7).[323]

Prognosis

One of the most difficult challenges in CPR is to decide the point at which further resuscitative efforts are futile. Unfortunately, there are no simple guidelines. Certain characteristics suggest that resuscitation should be continued (e.g. ice water drowning, witnessed VF arrest), and others suggest that further resuscitative efforts will be futile (e.g. most cardiac arrests associated with blunt trauma or septic shock).

Predictors of outcome in children
W12B,W28

Consensus on science. Multiple studies in adults have linked characteristics of the patient or of the cardiac arrest with prognosis following in-hospital or out-of-hospital cardiac arrest. Experience in children is more limited. Six paediatric studies (LOE 5)[3,324—328] show that prolonged resuscitation is associated with a poor outcome. Although the likelihood of a good outcome is greater with a short duration of CPR, two paediatric studies (LOE 3)[328,329] reported good outcomes in some patients following 30—60 min of CPR in the in-patient setting when the arrests were witnessed and prompt and presumably excellent CPR was provided. Children with cardiac arrest associated with environmental hypothermia or immersion in icy water can have excellent outcomes despite >30 min of cardiac arrest (LOE 5).[7,330]

One large paediatric study (LOE 4)[331] and several smaller studies (LOE 5)[332–336] show that good outcome can be achieved when extracorporeal CPR is started after 30—90 min of refractory standard CPR for in-hospital cardiac arrests. The good outcomes were reported primarily in patients with isolated heart disease. These data show that 15 or 30 min of CPR does not define the limits of cardiac and cerebral viability.

Witnessed events, bystander CPR, and a short interval from collapse to arrival of EMS system personnel are important prognostic factors associated with improved outcome in adult resuscitation, and it seems reasonable to extrapolate these factors to children. At least one paediatric study (LOE 5)[328] showed that the interval from collapse to initiation of CPR is a significant prognostic factor.

Children with prehospital cardiac arrest caused by blunt trauma[337] and in-hospital cardiac arrest caused by septic shock[329] rarely survive.

Treatment recommendation. The rescuer should consider whether to discontinue resuscitative efforts after 15—20 min of CPR. Relevant considerations include the cause of the arrest, pre-existing conditions, whether the arrest was witnessed, duration of untreated cardiac arrest (no flow), effectiveness and duration of CPR (low flow), prompt availability of extracorporeal life support for a reversible disease process, and associated special circumstances (e.g. icy water drowning, toxic drug exposure).

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.resuscitation.2005.09.020.

References

1. American Heart Association in collaboration with International Liaison Committee on Resuscitation. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: International Consensus on Science, Part 9: Pediatric Basic Life Support. Resuscitation 2000;46:301—42.
2. American Heart Association in collaboration with International Liaison Committee on Resuscitation. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: International Consensus on Science, Part 10: Pediatric Advanced Life Support. Resuscitation 2000; 46:343—400.
3. Zaritsky A, Nadkarni V, Getson P, Kuehl K. CPR in children. Ann Emerg Med 1987;16:1107—11.
4. Hickey RW, Cohen DM, Strausbaugh S, Dietrich AM. Pediatric patients requiring CPR in the prehospital setting. Ann Emerg Med 1995;25:495—501.
5. Mogayzel C, Quan L, Graves JR, Tiedeman D, Fahrenbruch C, Herndon P. Out-of-hospital ventricular fibrillation in children and adolescents: causes and outcomes. Ann Emerg Med 1995;25:484—91.
6. Herlitz J, Engdahl J, Svensson L, Young M, Angquist KA, Holmberg S. Characteristics and outcome among children suffering from out of hospital cardiac arrest in Sweden. Resuscitation 2005;64:37—40.
7. Kuisma M, Suominen P, Korpela R. Paediatric out-of-hospital cardiac arrests: epidemiology and outcome. Resuscitation 1995;30:141—50.
8. Kyriacou DN, Arcinue EL, Peek C, Kraus JF. Effect of immediate resuscitation on children with submersion injury. Pediatrics 1994;94:137—42.
9. Berg RA, Hilwig RW, Kern KB, Ewy GA. ''Bystander'' chest compressions and assisted ventilation independently improve outcome from piglet asphyxial pulseless ''cardiac arrest''. Circulation 2000;101:1743—8.
10. Safranek DJ, Eisenberg MS, Larsen MP. The epidemiology of cardiac arrest in young adults. Ann Emerg Med 1992;21:1102—6.
11. Berg RA, Hilwig RW, Kern KB, Ewy GA. Precountershock cardiopulmonary resuscitation improves ventricular fibrillation median frequency and myocardial readiness for successful defibrillation from prolonged ventricular fibrillation: a randomized, controlled swine study. Ann Emerg Med 2002;40:563—70.
12. Berg RA, Hilwig RW, Kern KB, Sanders AB, Xavier LC, Ewy GA. Automated external defibrillation versus manual defibrillation for prolonged ventricular fibrillation: lethal delays of chest compressions before and after countershocks. Ann Emerg Med 2003;42:458—67.
13. Cobb LA, Fahrenbruch CE, Walsh TR, et al. Influence of cardiopulmonary resuscitation prior to defibrillation in patients with out-of-hospital ventricular fibrillation. JAMA 1999;281:1182—8.
14. Dowie R, Campbell H, Donohoe R, Clarke P. 'Event tree' analysis of out-of-hospital cardiac arrest data: confirming the importance of bystander CPR. Resuscitation 2003;56:173—81.
15. Engdahl J, Bang A, Lindqvist J, Herlitz J. Factors affecting short- and long-term prognosis among 1069 patients with out-of-hospital cardiac arrest and pulseless electrical activity. Resuscitation 2001;51:17—25.
16. Holmberg M, Holmberg S, Herlitz J. Effect of bystander cardiopulmonary resuscitation in out-of-hospital cardiac arrest patients in Sweden. Resuscitation 2000;47: 59—70.
17. Stiell IG, Nichol G, Wells G, et al. Health-related quality of life is better for cardiac arrest survivors who received citizen cardiopulmonary resuscitation. Circulation 2003;108:1939—44.
18. Eftestol T, Wik L, Sunde K, Steen PA. Effects of cardiopulmonary resuscitation on predictors of ventricular fibrillation defibrillation success during out-of-hospital cardiac arrest. Circulation 2004;110:10—5.
19. Wik L, Hansen TB, Fylling F, et al. Delaying defibrillation to give basic cardiopulmonary resuscitation to patients with out-of-hospital ventricular fibrillation: a randomized trial. JAMA 2003;289:1389—95.
20. Eberle B, Dick WF, Schneider T, Wisser G, Doetsch S, Tzanova I. Checking the carotid pulse check: diagnostic accuracy of first responders in patients with and without a pulse. Resuscitation 1996;33:107—16.

21. Owen CJ, Wyllie JP. Determination of heart rate in the baby at birth. Resuscitation 2004;60:213—7.
22. Bahr J, Klingler H, Panzer W, Rode H, Kettler D. Skills of lay people in checking the carotid pulse. Resuscitation 1997;35:23—6.
23. Cavallaro DL, Melker RJ. Comparison of two techniques for detecting cardiac activity in infants. Crit Care Med 1983;11:189—90.
24. Graham CA, Lewis NF. Evaluation of a new method for the carotid pulse check in cardiopulmonary resuscitation. Resuscitation 2002;53:37—40.
25. Lee CJ, Bullock LJ. Determining the pulse for infant CPR: time for a change? Mil Med 1991;156:190—3.
26. Ochoa FJ, Ramalle-Gomara E, Carpintero JM, Garcia A, Saralegui I. Competence of health professionals to check the carotid pulse. Resuscitation 1998;37:173—5.
27. Mather C, O'Kelly S. The palpation of pulses. Anaesthesia 1996;51:189—91.
28. Lapostolle F, Le Toumelin P, Agostinucci JM, Catineau J, Adnet F. Basic cardiac life support providers checking the carotid pulse: performance, degree of conviction, and influencing factors. Acad Emerg Med 2004;11:878—80.
29. Moule P. Checking the carotid pulse: diagnostic accuracy in students of the healthcare professions. Resuscitation 2000;44:195—201.
30. Bahr J. CPR education in the community. Eur J Emerg Med 1994;1:190—2.
31. Inagawa G, Morimura N, Miwa T, Okuda K, Hirata M, Hiroki K. A comparison of five techniques for detecting cardiac activity in infants. Paediatr Anaesth 2003;13:141—6.
32. Tanner M, Nagy S, Peat JK. Detection of infant's heart beat/pulse by caregivers: a comparison of 4 methods. J Pediatr 2000;137:429—30.
33. Tonkin SL, Gunn AJ. Failure of mouth-to-mouth resuscitation in cases of sudden infant death. Resuscitation 2001;48:181—4.
34. Wilson-Davis SL, Tonkin SL, Gunn TR. Air entry in infant resuscitation: oral or nasal routes? J Appl Physiol 1997;82:152—5.
35. Stocks J, Godfrey S. Nasal resistance during infancy. Respir Physiol 1978;34:233—46.
36. Rodenstein DO, Perlmutter N, Stanescu DC. Infants are not obligatory nasal breathers. Am Rev Respir Dis 1985;131:343—7.
37. James DS, Lambert WE, Mermier CM, Stidley CA, Chick TW, Samet JM. Oronasal distribution of ventilation at different ages. Arch Environ Health 1997;52:118—23.
38. Berg MD, Idris AH, Berg RA. Severe ventilatory compromise due to gastric distention during pediatric cardiopulmonary resuscitation. Resuscitation 1998;36:71—3.
39. Segedin E, Torrie J, Anderson B. Nasal airway versus oral route for infant resuscitation. Lancet 1995;346:382.
40. Dembofsky CA, Gibson E, Nadkarni V, Rubin S, Greenspan JS. Assessment of infant cardiopulmonary resuscitation rescue breathing technique: relationship of infant and caregiver facial measurements. Pediatrics 1999;103:E17.
41. Tonkin SL, Davis SL, Gunn TR. Nasal route for infant resuscitation by mothers. Lancet 1995;345:1353—4.
42. Sorribes del Castillo J, Carrion Perez C, Sanz Ribera J. Nasal route to ventilation during basic cardiopulmonary resuscitation in children under two months of age. Resuscitation 1997;35:249—52.
43. Nowak AJ, Casamassimo PS. Oral opening and other selected facial dimensions of children 6 weeks to 36 months of age. J Oral Maxillofac Surg 1994;52:845—7 [discussion 8].
44. Whitelaw CC, Slywka B, Goldsmith LJ. Comparison of a two-finger versus two-thumb method for chest compressions by healthcare providers in an infant mechanical model. Resuscitation 2000;43:213—6.
45. Dorfsman ML, Menegazzi JJ, Wadas RJ, Auble TE. Two-thumb vs two-finger chest compression in an infant model of prolonged cardiopulmonary resuscitation. Acad Emerg Med 2000;7:1077—82.
46. Menegazzi JJ, Auble TE, Nicklas KA, Hosack GM, Rack L, Goode JS. Two-thumb versus two-finger chest compression during CRP in a swine infant model of cardiac arrest. Ann Emerg Med 1993;22:240—3.
47. Houri PK, Frank LR, Menegazzi JJ, Taylor R. A randomized, controlled trial of two-thumb vs two-finger chest compression in a swine infant model of cardiac arrest. Prehosp Emerg Care 1997;1:65—7.
48. Todres ID, Rogers MC. Methods of external cardiac massage in the newborn infant. J Pediatr 1975;86:781—2.
49. David R. Closed chest cardiac massage in the newborn infant. Pediatrics 1988;81:552—4.
50. Stevenson AG, McGowan J, Evans AL, Graham CA. CPR for children: one hand or two? Resuscitation 2005;64:205—8.
51. Kinney SB, Tibballs J. An analysis of the efficacy of bag-valve-mask ventilation and chest compression during different compression—ventilation ratios in manikin-simulated paediatric resuscitation. Resuscitation 2000;43:115—20.
52. Dorph E, Wik L, Steen PA. Effectiveness of ventilation-compression ratios 1:5 and 2:15 in simulated single rescuer paediatric resuscitation. Resuscitation 2002;54:259—64.
53. Greingor JL. Quality of cardiac massage with ratio compression—ventilation 5/1 and 15/2. Resuscitation 2002;55:263—7.
54. Srikantan S, Berg RA, Cox T, Tice L, Nadkarni VM. Effect of one-rescuer compression/ventilation ratios on cardiopulmonary resuscitation in infant, pediatric, and adult manikins. Pediatr Crit Care Med 2005;6:293—7.
55. Babbs CF, Nadkarni V. Optimizing chest compression to rescue ventilation ratios during one-rescuer CPR by professionals and lay persons: children are not just little adults. Resuscitation 2004;61:173—81.
56. Berg RA, Sanders AB, Kern KB, et al. Adverse hemodynamic effects of interrupting chest compressions for rescue breathing during cardiopulmonary resuscitation for ventricular fibrillation cardiac arrest. Circulation 2001;104:2465—70.
57. Kern KB, Hilwig RW, Berg RA, Ewy GA. Efficacy of chest compression-only BLS CPR in the presence of an occluded airway. Resuscitation 1998;39:179—88.
58. Assar D, Chamberlain D, Colquhoun M, et al. Randomised controlled trials of staged teaching for basic life support, 1: skill acquisition at bronze stage. Resuscitation 2000;45:7—15.
59. Heidenreich JW, Higdon TA, Kern KB, et al. Single-rescuer cardiopulmonary resuscitation: 'two quick breaths'—an oxymoron. Resuscitation 2004;62:283—9.
60. Abella BS, Alvarado JP, Myklebust H, et al. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. JAMA 2005;293:305—10.
61. Wik L, Kramer-Johansen J, Myklebust H, et al. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. JAMA 2005;293:299—304.
62. Eftestol T, Sunde K, Steen PA. Effects of interrupting precordial compressions on the calculated probability of defibrillation success during out-of-hospital cardiac arrest. Circulation 2002;105:2270—3.

63. Yu T, Weil MH, Tang W, et al. Adverse outcomes of interrupted precordial compression during automated defibrillation. Circulation 2002;106:368—72.
64. Abella BS, Sandbo N, Vassilatos P, et al. Chest compression rates during cardiopulmonary resuscitation are suboptimal: a prospective study during in-hospital cardiac arrest. Circulation 2005;111:428—34.
65. Berg RA, Hilwig RW, Kern KB, Babar I, Ewy GA. Simulated mouth-to-mouth ventilation and chest compressions (bystander cardiopulmonary resuscitation) improves outcome in a swine model of prehospital pediatric asphyxial cardiac arrest. Crit Care Med 1999;27:1893—9.
66. Kern KB, Hilwig RW, Berg RA, Sanders AB, Ewy GA. Importance of continuous chest compressions during cardiopulmonary resuscitation: improved outcome during a simulated single lay-rescuer scenario. Circulation 2002;105:645—9.
67. Becker LB, Berg RA, Pepe PE, et al. A reappraisal of mouth-to-mouth ventilation during bystander-initiated cardiopulmonary resuscitation. A statement for healthcare professionals from the Ventilation Working Group of the Basic Life Support and Pediatric Life Support Subcommittees, American Heart Association. Resuscitation 1997;35:189—201.
68. Suominen P, Rasanen J, Kivioja A. Efficacy of cardiopulmonary resuscitation in pulseless paediatric trauma patients. Resuscitation 1998;36:9—13.
69. Christensen DW, Jansen P, Perkin RM. Outcome and acute care hospital costs after warm water near drowning in children. Pediatrics 1997;99:715—21.
70. Young KD, Seidel JS. Pediatric cardiopulmonary resuscitation: a collective review. Ann Emerg Med 1999;33:195—205.
71. Waalewijn RA, Tijssen JG, Koster RW. Bystander initiated actions in out-of-hospital cardiopulmonary resuscitation: results from the Amsterdam Resuscitation Study (ARRESUST). Resuscitation 2001;50:273—9.
72. Holmberg M, Holmberg S, Herlitz J. Factors modifying the effect of bystander cardiopulmonary resuscitation on survival in out-of-hospital cardiac arrest patients in Sweden. Eur Heart J 2001;22:511—9.
73. Bossaert L, Van Hoeyweghen R, The Cerebral Resuscitation Study Group. Bystander cardiopulmonary resuscitation (CPR) in out-of-hospital cardiac arrest. Resuscitation 1989;17(Suppl.):S55—69.
74. Hallstrom A, Cobb L, Johnson E, Copass M. Cardiopulmonary resuscitation by chest compression alone or with mouth-to-mouth ventilation. N Engl J Med 2000;342:1546—53.
75. Berg RA, Kern KB, Sanders AB, Otto CW, Hilwig RW, Ewy GA. Bystander cardiopulmonary resuscitation. Is ventilation necessary? Circulation 1993;88:1907—15.
76. Berg RA, Wilcoxson D, Hilwig RW, et al. The need for ventilatory support during bystander CPR. Ann Emerg Med 1995;26:342—50.
77. Berg RA, Kern KB, Hilwig RW, et al. Assisted ventilation does not improve outcome in a porcine model of single-rescuer bystander cardiopulmonary resuscitation. Circulation 1997;95:1635—41.
78. Berg RA, Kern KB, Hilwig RW, Ewy GA. Assisted ventilation during 'bystander' CPR in a swine acute myocardial infarction model does not improve outcome. Circulation 1997;96:4364—71.
79. Engoren M, Plewa MC, Buderer NF, Hymel G, Brookfield L. Effects of simulated mouth-to-mouth ventilation during external cardiac compression or active compression-decompression in a swine model of witnessed cardiac arrest. Ann Emerg Med 1997;29:607—15.
80. Wen ZC, Chen SA, Tai CT, Chiang CE, Chiou CW, Chang MS. Electrophysiological mechanisms and determinants of vagal maneuvers for termination of paroxysmal supraventricular tachycardia. Circulation 1998;98:2716—23.
81. Bisset GSI, Gaum W, Kaplan S. The ice bag: a new technique for interruption of supraventricular tachycardia. J Pediatr 1980;97:593—5.
82. Sreeram N, Wren C. Supraventricular tachycardia in infants: response to initial treatment. Arch Dis Child 1990;65:127—9.
83. Mehta D, Wafa S, Ward DE, Camm AJ. Relative efficacy of various physical manoeuvres in the termination of junctional tachycardia. Lancet 1988;1:1181—5.
84. Lim SH, Anantharaman V, Teo WS, Goh PP, Tan AT. Comparison of treatment of supraventricular tachycardia by Valsalva maneuver and carotid sinus massage. Ann Emerg Med 1998;31:30—5.
85. Josephson ME, Seides SE, Batsford WB, Caracta AR, Damato AN, Kastor JA. The effects of carotid sinus pressure in re-entrant paroxysmal supraventricular tachycardia. Am Heart J 1974;88:694—7.
86. Waxman MB, Wald RW, Sharma AD, Huerta F, Cameron DA. Vagal techniques for termination of paroxysmal supraventricular tachycardia. Am J Cardiol 1980;46:655—64.
87. Wayne MA. Conversion of paroxysmal atrial tachycardia by facial immersion in ice water. JACEP 1976;5:434—5.
88. Wildenthal K, Leshin SJ, Atkins JM, Skelton CL. The diving reflex used to treat paroxysmal atrial tachycardia. Lancet 1975;1:12—4.
89. Hamilton J, Moodie D, Levy J. The use of the diving reflex to terminate supraventricular tachycardia in a 2-week-old infant. Am Heart J 1979;97:371—4.
90. Craig JE, Scholz TA, Vanderhooft SL, Etheridge SP. Fat necrosis after ice application for supraventricular tachycardia termination. J Pediatr 1998;133:727.
91. Thomas MD, Torres A, Garcia-Polo J, Gavilan C. Life-threatening cervico-mediastinal haematoma after carotid sinus massage. J Laryngol Otol 1991;105:381—3.
92. Bianconi L, Castro A, Dinelli M, et al. Comparison of intravenously administered dofetilide versus amiodarone in the acute termination of atrial fibrillation and flutter. A multicentre, randomized, double-blind, placebo-controlled study. Eur Heart J 2000; 21:1265—73.
93. Burri S, Hug MI, Bauersfeld U. Efficacy and safety of intravenous amiodarone for incessant tachycardias in infants. Eur J Pediatr 2003;162:880—4.
94. Cabrera Duro A, Rodrigo Carbonero D, Galdeano Miranda J, et al. The treatment of postoperative junctional ectopic tachycardia, Spanish. Anales Espanoles de Pediatria 2002;56:505—9.
95. Celiker A, Ceviz N, Ozme S. Effectiveness and safety of intravenous amiodarone in drug-resistant tachyarrhythmias of children. Acta Paediatrica Japonica 1998;40:567—72.
96. Dodge-Khatami A, Miller O, Anderson R, Gil-Jaurena J, Goldman A, de Leval M. Impact of junctional ectopic tachycardia on postoperative morbidity following repair of congenital heart defects. Euro J Cardio-Thorac Surg 2002;21:255—9.
97. Figa FH, Gow RM, Hamilton RM, Freedom RM. Clinical efficacy and safety of intravenous Amiodarone in infants and children. Am J Cardiol 1994;74:573—7.
98. Hoffman TM, Bush DM, Wernovsky G, et al. Postoperative junctional ectopic tachycardia in children: incidence, risk factors, and treatment. Ann Thorac Surg 2002;74:1607—11.
99. Laird WP, Snyder CS, Kertesz NJ, Friedman RA, Miller D, Fenrich AL. Use of intravenous amiodarone for postoper-

ative junctional ectopic tachycardia in children. Pediatr Cardiol 2003;24:133—7.
100. Perry JC, Fenrich AL, Hulse JE, Triedman JK, Friedman RA, Lamberti JJ. Pediatric use of intravenous amiodarone: efficacy and safety in critically ill patients from a multicenter protocol. J Am Coll Cardiol 1996;27:1246—50.
101. Soult JA, Munoz M, Lopez JD, Romero A, Santos J, Tovaruela A. Efficacy and safety of intravenous amiodarone for short-term treatment of paroxysmal supraventricular tachycardia in children. Pediatr Cardiol 1995;16:16—9.
102. Valsangiacomo Eea. Early postoperative arrhythmias after cardiac operation in children. Ann Thorac Surg 2002;72:792—6.
103. Yap S-C, Hoomtje T, Sreeram N. Polymorphic ventricular tachycardia after use of intravenous amiodarone for postoperative junctional ectopic tachycardia. Int J Cardiol 2000;76:245—7.
104. Daniels CJ, Schutte DA, Hammond S, Franklin WH. Acute pulmonary toxicity in an infant from intravenous amiodarone. Am J Cardiol 1998;80:1113—6.
105. Gandy J, Wonko N, Kantoch MJ. Risks of intravenous amiodarone in neonates. Can J Cardiol 1998;14:855—8.
106. Benson DJ, Dunnigan A, Green T, Benditt D, Schneider S. Periodic procainamide for paroxysmal tachycardia. Circulation 1985;72:147—52.
107. Boahene KA, Klein GJ, Yee R, Sharma AD, Fujimura O. Termination of acute atrial fibrillation in the Wolff-Parkinson-White syndrome by procainamide and propafenone: importance of atrial fibrillatory cycle length. J Am Coll Cardiol 1990;16:1408—14.
108. Dodo H, Gow RM, Hamilton RM, Freedom RM. Chaotic atrial rhythm in children. Am Heart J 1995;129:990—5.
109. Komatsu C, Ishinaga T, Tateishi O, Tokuhisa Y, Yoshimura S. Effects of four antiarrhythmic drugs on the induction and termination of paroxysmal supraventricular tachycardia. Jpn Circ J 1986;50:961—72.
110. Mandapati R, Byrum CJ, Kavey RE, et al. Procainamide for rate control of postsurgical junctional tachycardia. Pediatr Cardiol 2000;21:123—8.
111. Mandel WJ, Laks MM, Obayashi K, Hayakawa H, Daley W. The Wolff-Parkinson-White syndrome: pharmacologic effects of procaine amide. Am Heart J 1975;90:744—54.
112. Mehta AV, Sanchez GR, Sacks EJ, Casta A, Dunn JM, Donner RM. Ectopic automatic atrial tachycardia in children: clinical characteristics, management and follow-up. J Am Coll Cardiol 1988;11:379—85.
113. Rhodes LA, Walsh EP, Saul JP. Conversion of atrial flutter in pediatric patients by transesophageal atrial pacing: a safe, effective, minimally invasive procedure. Am Heart J 1995;130:323—7.
114. Satake S, Hiejima K, Moroi Y, Hirao K, Kubo I, Suzuki F. Usefulness of invasive and non-invasive electrophysiologic studies in the selection of antiarrhythmic drugs for the patients with paroxysmal supraventricular tachyarrhythmia. Jpn Circ J 1985;49:345—50.
115. Singh S, Gelband H, Mehta A, Kessler K, Casta A, Pickoff A. Procainamide elimination kinetics in pediatric patients. Clin Pharmacol Therap 1982;32:607—11.
116. Walsh EP, Saul JP, Sholler GF, et al. Evaluation of a staged treatment protocol for rapid automatic junctional tachycardia after operation for congenital heart disease. J Am Coll Cardiol 1997;29:1046—53.
117. Wang JN, Wu JM, Tsai YC, Lin CS. Ectopic atrial tachycardia in children. J Formos Med Assoc 2000;99:766—70.
118. Chen F, Wetzel G, Klitzner TS. Acute effects of amiodarone on sodium currents in isolated neonatal ventricular myocytes: comparison with procainamide. Develop Pharmacol Therap 1992;19:118—30.
119. Fujiki A, Tani M, Yoshida S, Inoue H. Electrophysiologic mechanisms of adverse effects of class I antiarrhythmic drugs (cibenzoline, pilsicainide, disopyramide, procainamide) in induction of atrioventricular re-entrant tachycardia. Cardiovasc Drug Ther 1996;10:159—66.
120. Bauernfeind RA, Swiryn S, Petropoulos AT, Coelho A, Gallastegui J, Rosen KM. Concordance and discordance of drug responses in atrioventricular reentrant tachycardia. J Am Coll Cardiol 1983;2:345—50.
121. Hordof AJ, Edie R, Malm JR, Hoffman BF, Rosen MR. Electrophysiologic properties and response to pharmacologic agents of fibers from diseased human atria. Circulation 1976;54:774—9.
122. Jawad-Kanber G, Sherrod TR. Effect of loading dose of procaine amide on left ventricular performance in man. Chest 1974;66:269—72.
123. Karlsson E, Sonnhag C. Haemodynamic effects of procainamide and phenytoin at apparent therapeutic plasma levels. Eur J Clin Pharmacol 1976;10:305—10.
124. Shih JY, Gillette PC, Kugler JD, et al. The electrophysiologic effects of procainamide in the immature heart. Pediatr Pharmacol (New York) 1982;2:65—73.
125. Benson Jr D, Smith W, Dunnigan A, Sterba R, Gallagher J. Mechanisms of regular wide QRS tachycardia in infants and children. Am J Cardiol 1982;49:1778—88.
126. Kuga K, Yamaguchi I, Sugishita Y. Effect of intravenous amiodarone on electrophysiologic variables and on the modes of termination of atrioventricular reciprocating tachycardia in Wolff-Parkinson-White syndrome. Jpn Circ J 1999;63:189—95.
127. Juneja R, Shah S, Naik N, Kothari SS, Saxena A, Talwar KK. Management of cardiomyopathy resulting from incessant supraventricular tachycardia in infants and children. Indian Heart J 2002;54:176—80.
128. Michael JG, Wilson Jr WR, Tobias JD. Amiodarone in the treatment of junctional ectopic tachycardia after cardiac surgery in children: report of two cases and review of the literature. Am J Ther 1999;6:223—7.
129. Perry JC, Knilans TK, Marlow D, Denfield SW, Fenrich AL, Friedman RA. Intravenous amiodarone for life-threatening tachyarrhythmias in children and young adults. J Am Coll Cardiol 1993;22:95—8.
130. Singh BN, Kehoe R, Woosley RL, Scheinman M, Quart B, Sotalol Multicenter Study Group. Multicenter trial of sotalol compared with procainamide in the suppression of inducible ventricular tachycardia: a double-blind, randomized parallel evaluation. Am Heart J 1995;129:87—97.
131. Meldon SW, Brady WJ, Berger S, Mannenbach M. Pediatric ventricular tachycardia: a review with three illustrative cases. Pediatr Emerg Care 1994;10:294—300.
132. Stanton MS, Prystowsky EN, Fineberg NS, Miles WM, Zipes DP, Heger JJ. Arrhythmogenic effects of antiarrhythmic drugs: a study of 506 patients treated for ventricular tachycardia or fibrillation. J Am Coll Cardiol 1989;14:209—15 [discussion 16—7].
133. Hasin Y, Kriwisky M, Gotsman MS. Verapamil in ventricular tachycardia. Cardiology 1984;71:199—206.
134. Hernandez A, Strauss A, Kleiger RE, Goldring D. Idiopathic paroxysmal ventricular tachycardia in infants and children. J Pediatr 1975;86:182—8.
135. Horowitz LN, Josephson ME, Farshidi A, Spielman SR, Michelson EL, Greenspan AM. Recurrent sustained ventricular tachycardia 3. Role of the electrophysiologic study in selection of antiarrhythmic regimens. Circulation 1978;58:986—97.

136. Mason JW, Winkle RA. Electrode-catheter arrhythmia induction in the selection and assessment of antiarrhythmic drug therapy for recurrent ventricular tachycardia. Circulation 1978;58:971—85.
137. Cain ME, Martin TC, Marchlinski FE, Josephson ME. Changes in ventricular refractoriness after an extrastimulus: effects of prematurity, cycle length and procainamide. Am J Cardiol 1983;52:996—1001.
138. Swiryn S, Bauernfeind RA, Strasberg B, et al. Prediction of response to class I antiarrhythmic drugs during electrophysiologic study of ventricular tachycardia. Am Heart J 1982;104:43—50.
139. Velebit V, Podrid P, Lown B, Cohen B, Graboys T. Aggravation and provocation of ventricular arrhythmias by antiarrhythmic drugs. Circulation 1982;65:886—94.
140. Roden D, Reele S, Higgins S, et al. Antiarrhythmic efficacy, pharmacokinetics and safety of *N*-acetylprocainamide in human subjects: comparison with procainamide. Am J Cardiol 1980;46:463—8.
141. Naitoh N, Washizuka T, Takahashi K, Aizawa Y. Effects of class I and III antiarrhythmic drugs on ventricular tachycardia-interrupting critical paced cycle length with rapid pacing. Jpn Circ J 1998;62:267—73.
142. Kasanuki H, Ohnishi S, Hosoda S. Differentiation and mechanisms of prevention and termination of verapamil-sensitive sustained ventricular tachycardia. Am J Cardiol 1989;64:46J—9J.
143. Kasanuki H, Ohnishi S, Tanaka E, Hirosawa K. Clinical significance of Vanghan Williams classification for treatment of ventricular tachycardia: study of class IA and IB antiarrhythmic agents. Jpn Circ J 1988;52:280—8.
144. Videbaek J, Andersen E, Jacobsen J, Sandoe E, Wennevold A. Paroxysmal tachycardia in infancy and childhood. II. Paroxysmal ventricular tachycardia and fibrillation. Acta Paediatrica Scandinavica 1973;62:349—57.
145. Sanchez J, Christie K, Cumming G. Treatment of ventricular tachycardia in an infant. Can Med Assoc J 1972;107:136—8.
146. Gelband H, Steeg C, Bigger JJ. Use of massive doses of procaineamide in the treatment of ventricular tachycardia in infancy. Pediatrics 1971;48:110—5.
147. Drago F, Mazza A, Guccione P, Mafrici A, Di Liso G, Ragonese P. Amiodarone used alone or in combination with propranolol: a very effective therapy for tachyarrhythmias in infants and children. Pediatr Cardiol 1998;19:445—9.
148. Rokicki W, Durmala J, Nowakowska E. Amiodarone for long term treatment of arrhythmia in children. Wiad Lek 2001;54:45—50.
149. Strasburger JF, Cuneo BF, Michon MM, et al. Amiodarone therapy for drug-refractory fetal tachycardia. Circulation 2004;109:375—9.
150. Beder SD, Cohen MH, BenShachar G. Time course of myocardial amiodarone uptake in the piglet heart using a chronic animal model. Pediatr Cardiol 1998;19:204—11.
151. Paiva EF, Perondi MB, Kern KB, et al. Effect of amiodarone on haemodynamics during cardiopulmonary resuscitation in a canine model of resistant ventricular fibrillation. Resuscitation 2003;58:203—8.
152. Aiba T, Kurita T, Taguchi A, et al. Long-term efficacy of empirical chronic amiodarone therapy in patients with sustained ventricular tachyarrhythmia and structural heart disease. Circ J 2002;66:367—71.
153. Cairns JA, Connolly SJ, Gent M, Roberts R. Post-myocardial infarction mortality in patients with ventricular premature depolarisations. Canadian Amiodarone Myocardial Infarction Arrhythmia Trial Pilot Study. Circulation 1991;84:550—7.
154. Dorian P, Cass D, Schwartz B, Cooper R, Gelaznikas R, Barr A. Amiodarone as compared with lidocaine for shock-resistant ventricular fibrillation. N Engl J Med 2002;346:884—90.
155. Fogel RI, Herre JM, Kopelman HA, et al. Long-term follow-up of patients requiring intravenous amiodarone to suppress hemodynamically destabilizing ventricular arrhythmias. Am Heart J 2000;139:690—5.
156. Heger JJ, Prystowsky EN, Jackman WM, et al. Clinical efficacy and electrophysiology during long-term therapy for recurrent ventricular tachycardia or ventricular fibrillation. N Engl J Med 1981;305:539—45.
157. Kalbfleisch SJ, Williamson B, Man KC, et al. Prospective, randomized comparison of conventional and high dose loading regimens of amiodarone in the treatment of ventricular tachycardia. J Am Coll Cardiol 1993;22:1723—9.
158. Kowey PR, Levine JH, Herre JM, et al. Randomized, double-blind comparison of intravenous amiodarone and bretylium in the treatment of patients with recurrent, hemodynamically destabilizing ventricular tachycardia or fibrillation. The Intravenous Amiodarone Multicenter Investigators Group. Circulation 1995;92:3255—63.
159. Kudenchuk PJ, Cobb LA, Copass MK, et al. Amiodarone for resuscitation after out-of-hospital cardiac arrest due to ventricular fibrillation. N Engl J Med 1999;341:871—8.
160. Lee KL, Tai YT. Long-term low-dose amiodarone therapy in the management of ventricular and supraventricular tachyarrhythmias: efficacy and safety. Clin Cardiol 1997;20:372—7.
161. Levine JH, Massumi A, Scheinman MM, et al. Intravenous amiodarone for recurrent sustained hypotensive ventricular tachyarrhythmias. Intravenous Amiodarone Multicenter Trial Group. J Am Coll Cardiol 1996;27:67—75.
162. Singh SN, Fletcher RD, Fisher SG, et al. Amiodarone in patients with congestive heart failure and asymptomatic ventricular arrhythmia. Survival Trial of Antiarrhythmic Therapy in Congestive Heart Failure. N Engl J Med 1995;333:77—82.
163. Somberg JC, Timar S, Bailin SJ, et al. Lack of a hypotensive effect with rapid administration of a new aqueous formulation of intravenous amiodarone. Am J Cardiol 2004;93:576—81.
164. Sim I, McDonald KM, Lavori PW, Norbutas CM, Hlatky MA. Quantitative overview of randomized trials of amiodarone to prevent sudden cardiac death. Circulation 1997;96:2823—9.
165. Scheinman MM, Levine JH, Cannom DS, et al. Dose-ranging study of intravenous amiodarone in patients with life-threatening ventricular tachyarrhythmias. The Intravenous Amiodarone Multicenter Investigators Group. Circulation 1995;92:3264—72.
166. Schneider T, Martens PR, Paschen H, et al. Multicenter, randomized, controlled trial of 150-J biphasic shocks compared with 200- to 360-J monophasic shocks in the resuscitation of out-of-hospital cardiac arrest victims. Optimized Response to Cardiac Arrest (ORCA) Investigators. Circulation 2000;102:1780—7.
167. van Alem AP, Chapman FW, Lank P, Hart AA, Koster RW. A prospective, randomised and blinded comparison of first shock success of monophasic and biphasic waveforms in out-of-hospital cardiac arrest. Resuscitation 2003;58:17—24.
168. Higgins SL, Herre JM, Epstein AE, et al. A comparison of biphasic and monophasic shocks for external defibrillation. Physio-Control Biphasic Investigators. Prehosp Emerg Care 2000;4:305—13.

169. Carpenter J, Rea TD, Murray JA, Kudenchuk PJ, Eisenberg MS. Defibrillation waveform and post-shock rhythm in out-of-hospital ventricular fibrillation cardiac arrest. Resuscitation 2003;59:189—96.
170. Morrison LJ, Dorian P, Long J, et al. Out-of-hospital cardiac arrest rectilinear biphasic to monophasic damped sine defibrillation waveforms with advanced life support intervention trial (ORBIT). Resuscitation 2005;66:149—57.
171. Berg RA, Chapman FW, Berg MD, et al. Attenuated adult biphasic shocks compared with weight-based monophasic shocks in a swine model of prolonged pediatric ventricular fibrillation. Resuscitation 2004;61:189—97.
172. Clark CB, Zhang Y, Davies LR, Karlsson G, Kerber RE. Pediatric transthoracic defibrillation: biphasic versus monophasic waveforms in an experimental model. Resuscitation 2001;51:159—63.
173. Tang W, Weil MH, Jorgenson D, et al. Fixed-energy biphasic waveform defibrillation in a pediatric model of cardiac arrest and resuscitation. Crit Care Med 2002;30:2736—41.
174. Gutgesell HP, Tacker WA, Geddes LA, Davis S, Lie JT, McNamara DG. Energy dose for ventricular defibrillation of children. Pediatrics 1976;58:898—901.
175. Gurnett CA, Atkins DL. Successful use of a biphasic waveform automated external defibrillator in a high-risk child. Am J Cardiol 2000;86:1051—3.
176. Atkins D, Jorgenson D. Attenuated pediatric electrode pads for automated external defibrillator use in children. Resuscitation 2005;66:31—7.
177. Gaba DM, Talner NS. Myocardial damage following transthoracic direct current countershock in newborn piglets. Pediatr Cardiol 1982;2:281—8.
178. Killingsworth CR, Melnick SB, Chapman FW, et al. Defibrillation threshold and cardiac responses using an external biphasic defibrillator with pediatric and adult adhesive patches in pediatric-sized piglets. Resuscitation 2002;55:177—85.
179. Berg RA, Samson RA, Berg MD, et al. Better outcome after pediatric defibrillation dosage than adult dosage in a swine model of pediatric ventricular fibrillation. J Am Coll Cardiol 2005;45:786—9.
180. Berg RA, Hilwig RW, Ewy GA, Kern KB. Precountershock cardiopulmonary resuscitation improves initial response to defibrillation from prolonged ventricular fibrillation: a randomized, controlled swine study. Crit Care Med 2004;32:1352—7.
181. Somberg JC, Bailin SJ, Haffajee CI, et al. Intravenous lidocaine versus intravenous amiodarone (in a new aqueous formulation) for incessant ventricular tachycardia. Am J Cardiol 2002;90:853—9.
182. Gausche M, Lewis RJ, Stratton SJ, et al. Effect of out-of-hospital pediatric endotracheal intubation on survival and neurological outcome: a controlled clinical trial. JAMA 2000;283:783—90.
183. Pitetti R, Glustein JZ, Bhende MS. Prehospital care and outcome of pediatric out-of-hospital cardiac arrest. Prehosp Emerg Care 2002;6:283—90.
184. Cooper A, DiScala C, Foltin G, Tunik M, Markenson D, Welborn C. Prehospital endotracheal intubation for severe head injury in children: a reappraisal. Semin Pediatr Surg 2001;10:3—6.
185. Eckstein M, Chan L, Schneir A, Palmer R. Effect of prehospital advanced life support on outcomes of major trauma patients. J Trauma 2000;48:643—8.
186. Stockinger ZT, McSwain Jr NE. Prehospital endotracheal intubation for trauma does not improve survival over bag-valve-mask ventilation. J Trauma 2004;56:531—6.
187. Perron AD, Sing RF, Branas CC, Huynh T. Predicting survival in pediatric trauma patients receiving cardiopulmonary resuscitation in the prehospital setting. Prehosp Emerg Care 2001;5:6—9.
188. Khine HH, Corddry DH, Kettrick RG, et al. Comparison of cuffed and uncuffed endotracheal tubes in young children during general anesthesia. Anesthesiology 1997;86:627—31 [discussion 27A].
189. Newth CJ, Rachman B, Patel N, Hammer J. The use of cuffed versus uncuffed endotracheal tubes in pediatric intensive care. J Pediatr 2004;144:333—7.
190. Deakers TW, Reynolds G, Stretton M, Newth CJ. Cuffed endotracheal tubes in pediatric intensive care. J Pediatr 1994;125:57—62.
191. Bordet F, Allaouchiche B, Lansiaux S, et al. Risk factors for airway complications during general anaesthesia in paediatric patients. Paediatr Anaesth 2002;12:762—9.
192. Mhanna MJ, Zamel YB, Tichy CM, Super DM. The ''air leak'' test around the endotracheal tube, as a predictor of postextubation stridor, is age dependent in children. Crit Care Med 2002;30:2639—43.
193. Browning DH, Graves SA. Incidence of aspiration with endotracheal tubes in children. J Pediatr 1983;102:582—4.
194. Park C, Bahk JH, Ahn WS, Do SH, Lee KH. The laryngeal mask airway in infants and children. Can J Anaesth 2001;48:413—7.
195. Lopez-Gil M, Brimacombe J, Cebrian J, Arranz J. Laryngeal mask airway in pediatric practice: a prospective study of skill acquisition by anesthesia residents. Anesthesiology 1996;84:807—11.
196. Andersen KH, Hald A. Assessing the position of the tracheal tube: the reliability of different methods. Anaesthesia 1989;44:984—5.
197. Bhende MS, Thompson AE, Cook DR, Saville AL. Validity of a disposable end-tidal CO_2 detector in verifying endotracheal tube placement in infants and children. Ann Emerg Med 1992;21:142—5.
198. Bhende MS, Karasic DG, Karasic RB. End-tidal carbon dioxide changes during cardiopulmonary resuscitation after experimental asphyxial cardiac arrest. Am J Emerg Med 1996;14:349—50.
199. Kelly JS, Wilhoit RD, Brown RE, James R. Efficacy of the FEF colorimetric end-tidal carbon dioxide detector in children. Anesth Analg 1992;75:45—50.
200. Sharieff GQ, Rodarte A, Wilton N, Bleyle D. The self-inflating bulb as an airway adjunct: is it reliable in children weighing less than 20 kg? Acad Emerg Med 2003;10:303—8.
201. Bechtel K, Bhende M, Venkataraman S, Allen J. Use of esophageal detector device in a newborn-piglet model. Ann Emerg Med 1998;31:344—50.
202. Gausche-Hill M, Lewis RJ, Gunter CS, Henderson DP, Haynes BE, Stratton SJ. Design and implementation of a controlled trial of pediatric endotracheal intubation in the out-of-hospital setting. Ann Emerg Med 2000;36:356—65.
203. Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. Ann Emerg Med 2001;37:32—7.
204. Bhende MS, Allen Jr WD. Evaluation of a Capno-Flo resuscitator during transport of critically ill children. Pediatr Emerg Care 2002;18:414—6.
205. Campbell RC, Boyd CR, Shields RO, Odom JW, Corse KM. Evaluation of an end-tidal carbon dioxide detector in the aeromedical setting. J Air Med Transp 1990;9:13—5.
206. Gonzalez del Rey JA, Poirier MP, Digiulio GA. Evaluation of an ambu-bag valve with a self-contained, colorimetric end-tidal CO_2 system in the detection of airway mishaps: an animal trial. Pediatr Emerg Care 2000;16:121—3.

207. Poirier MP, Gonzalez Del-Rey JA, McAneney CM, DiGiulio GA. Utility of monitoring capnography, pulse oximetry, and vital signs in the detection of airway mishaps: a hyperoxemic animal model. Am J Emerg Med 1998;16:350—2.
208. Tan A, Schulze A, O'Donnell CP, Davis PG. Air versus oxygen for resuscitation of infants at birth. Cochrane Database Syst Rev 2004:CD002273.
209. Davis PG, Tan A, O'Donnell CP, Schulze A. Resuscitation of newborn infants with 100% oxygen or air: a systematic review and meta-analysis. Lancet 2004;364:1329—33.
210. Saugstad OD, Rootwelt T, Aalen O. Resuscitation of asphyxiated newborn infants with room air or oxygen: an international controlled trial: the Resair 2 study. Pediatrics 1998;102:e1.
211. Ramji S, Rasaily R, Mishra PK, et al. Resuscitation of asphyxiated newborns with room air or 100% oxygen at birth: a multicentric clinical trial. Indian Pediatr 2003;40:510—7.
212. Zwemer CF, Whitesall SE, D'Alecy LG. Cardiopulmonary-cerebral resuscitation with 100% oxygen exacerbates neurological dysfunction following nine minutes of normothermic cardiac arrest in dogs. Resuscitation 1994;27:159—70.
213. Liu Y, Rosenthal RE, Haywood Y, Miljkovic-Lolic M, Vanderhoek JY, Fiskum G. Normoxic ventilation after cardiac arrest reduces oxidation of brain lipids and improves neurological outcome. Stroke 1998;29:1679—86.
214. Lipinski CA, Hicks SD, Callaway CW. Normoxic ventilation during resuscitation and outcome from asphyxial cardiac arrest in rats. Resuscitation 1999;42:221—9.
215. Banerjee S, Singhi SC, Singh S, Singh M. The intraosseous route is a suitable alternative to intravenous route for fluid resuscitation in severely dehydrated children. Indian Pediatr 1994;31:1511—20.
216. Brickman KR, Krupp K, Rega P, Alexander J, Guinness M. Typing and screening of blood from intraosseous access. Ann Emerg Med 1992;21:414—7.
217. Fiser RT, Walker WM, Seibert JJ, McCarthy R, Fiser DH. Tibial length following intraosseous infusion: a prospective, radiographic analysis. Pediatr Emerg Care 1997;13:186—8.
218. Ummenhofer W, Frei FJ, Urwyler A, Drewe J. Are laboratory values in bone marrow aspirate predictable for venous blood in paediatric patients? Resuscitation 1994;27:123—8.
219. Glaeser PW, Hellmich TR, Szewczuga D, Losek JD, Smith DS. Five-year experience in prehospital intraosseous infusions in children and adults. Ann Emerg Med 1993;22:1119—24.
220. Guy J, Haley K, Zuspan SJ. Use of intraosseous infusion in the pediatric trauma patient. J Pediatr Surg 1993;28:158—61.
221. Macnab A, Christenson J, Findlay J, et al. A new system for sternal intraosseous infusion in adults. Prehosp Emerg Care 2000;4:173—7.
222. Ellemunter H, Simma B, Trawoger R, Maurer H. Intraosseous lines in preterm and full term neonates. Arch Dis Child Fetal Neonatal Ed 1999;80:F74—5.
223. Howard RF, Bingham RM. Endotracheal compared with intravenous administration of atropine. Arch Dis Child 1990;65:449—50.
224. Lee PL, Chung YT, Lee BY, Yeh CY, Lin SY, Chao CC. The optimal dose of atropine via the endotracheal route. Ma Zui Xue Za Zhi 1989;27:35—8.
225. Prengel AW, Lindner KH, Hahnel J, Ahnefeld FW. Endotracheal and endobronchial lidocaine administration: effects on plasma lidocaine concentration and blood gases. Crit Care Med 1991;19:911—5.
226. Schmidbauer S, Kneifel HA, Hallfeldt KK. Endobronchial application of high dose epinephrine in out of hospital cardiopulmonary resuscitation. Resuscitation 2000;47:89.
227. Raymondos K, Panning B, Leuwer M, et al. Absorption and hemodynamic effects of airway administration of adrenaline in patients with severe cardiac disease. Ann Intern Med 2000;132:800—3.
228. Hahnel JH, Lindner KH, Schurmann C, Prengel A, Ahnefeld FW. Plasma lidocaine levels and PaO_2 with endobronchial administration: dilution with normal saline or distilled water? Ann Emerg Med 1990;19:1314—7.
229. Brown LK, Diamond J. The efficacy of lidocaine in ventricular fibrillation due to coronary artery ligation: endotracheal vs intravenous use. Proc West Pharmacol Soc 1982;25: 43—5.
230. Jasani MS, Nadkarni VM, Finkelstein MS, Hofmann WT, Salzman SK. Inspiratory-cycle instillation of endotracheal epinephrine in porcine arrest. Acad Emerg Med 1994;1:340—5.
231. Wenzel V, Lindner KH, Prengel AW, Lurie KG, Strohmenger HU. Endobronchial vasopressin improves survival during cardiopulmonary resuscitation in pigs. Anesthesiology 1997;86:1375—81.
232. Vaknin Z, Manisterski Y, Ben-Abraham R, et al. Is endotracheal adrenaline deleterious because of the beta adrenergic effect? Anesth Analg 2001;92:1408—12.
233. Manisterski Y, Vaknin Z, Ben-Abraham R, et al. Endotracheal epinephrine: a call for larger doses. Anesth Analg 2002;95:1037—41 [table of contents].
234. Efrati O, Ben-Abraham R, Barak A, et al. Endobronchial adrenaline: should it be reconsidered? Dose response and haemodynamic effect in dogs. Resuscitation 2003;59:117—22.
235. Elizur A, Ben-Abraham R, Manisterski Y, et al. Tracheal epinephrine or norepinephrine preceded by beta blockade in a dog model. Can beta blockade bestow any benefits? Resuscitation 2003;59:271—6.
236. Perondi MB, Reis AG, Paiva EF, Nadkarni VM, Berg RA. A comparison of high-dose and standard-dose epinephrine in children with cardiac arrest. N Engl J Med 2004;350:1722—30.
237. Patterson MD, Boenning DA, Klein BL, et al. The use of high-dose epinephrine for patients with out-of-hospital cardiopulmonary arrest refractory to prehospital interventions. Pediatr Emerg Care 2005;21:227—37.
238. Carpenter TC, Stenmark KR. High-dose epinephrine is not superior to standard-dose epinephrine in pediatric in-hospital cardiopulmonary arrest. Pediatrics 1997;99: 403—8.
239. Dieckmann R, Vardis R. High-dose epinephrine in pediatric out-of-hospital cardiopulmonary arrest. Pediatrics 1995;95:901—13.
240. Mann K, Berg RA, Nadkarni V. Beneficial effects of vasopressin in prolonged pediatric cardiac arrest: a case series. Resuscitation 2002;52:149—56.
241. Voelckel WG, Lindner KH, Wenzel V, et al. Effects of vasopressin and epinephrine on splanchnic blood flow and renal function during and after cardiopulmonary resuscitation in pigs. Crit Care Med 2000;28:1083—8.
242. Voelckel WG, Lurie KG, McKnite S, et al. Effects of epinephrine and vasopressin in a piglet model of prolonged ventricular fibrillation and cardiopulmonary resuscitation. Crit Care Med 2002;30:957—62.
243. Wenzel V, Krismer AC, Arntz HR, Sitter H, Stadlbauer KH, Lindner KH. A comparison of vasopressin and epinephrine for out-of-hospital cardiopulmonary resuscitation. N Engl J Med 2004;350:105—13.
244. Stiell IG, Hebert PC, Wells GA, et al. Vasopressin versus epinephrine for inhospital cardiac arrest: a randomised controlled trial. Lancet 2001;358:105—9.

245. Lindner KH, Dirks B, Strohmenger HU, Prengel AW, Lindner IM, Lurie KG. Randomised comparison of epinephrine and vasopressin in patients with out-of-hospital ventricular fibrillation. Lancet 1997;349:535—7.
246. Guyette FX, Guimond GE, Hostler D, Callaway CW. Vasopressin administered with epinephrine is associated with a return of a pulse in out-of-hospital cardiac arrest. Resuscitation 2004;63:277—82.
247. Aung K, Htay T. Vasopressin for cardiac arrest: a systematic review and meta-analysis. Arch Intern Med 2005;165:17—24.
248. Cannon LA, Heiselman DE, Dougherty JM, Jones J. Magnesium levels in cardiac arrest victims: relationship between magnesium levels and successful resuscitation. Ann Emerg Med 1987;16:1195—9.
249. Buylaert WA, Calle PA, Houbrechts HN. Serum electrolyte disturbances in the post-resuscitation period. Resuscitation 1989;17:S189—96.
250. Salerno DM, Elsperger KJ, Helseth P, Murakami M, Chepuri V. Serum potassium, calcium and magnesium after resuscitation from ventricular fibrillation: a canine study. J Am Coll Cardiol 1987;10:178—85.
251. Allegra J, Lavery R, Cody R, et al. Magnesium sulfate in the treatment of refractory ventricular fibrillation in the prehospital setting. Resuscitation 2001;49:245—9.
252. Fatovich DM, Prentice DA, Dobb GJ. Magnesium in cardiac arrest (the magic trial). Resuscitation 1997;35:237—41.
253. Hassan TB, Jagger C, Barnett DB. A randomised trial to investigate the efficacy of magnesium sulphate for refractory ventricular fibrillation. Emerg Med J 2002;19:57—62.
254. Longstreth Jr WT, Fahrenbruch CE, Olsufka M, Walsh TR, Copass MK, Cobb LA. Randomized clinical trial of magnesium, diazepam, or both after out-of-hospital cardiac arrest. Neurology 2002;59:506—14.
255. Thel MC, Armstrong AL, McNulty SE, Califf RM, O'Connor CM. Randomised trial of magnesium in in-hospital cardiac arrest. Duke Internal Medicine Housestaff. Lancet 1997;350:1272—6.
256. Miller B, Craddock L, Hoffenberg S, et al. Pilot study of intravenous magnesium sulfate in refractory cardiac arrest: safety data and recommendations for future studies. Resuscitation 1995;30:3—14.
257. Brown CG, Griffith RF, Neely D, Hobson J, Miller B. The effect of intravenous magnesium administration on aortic, right atrial and coronary perfusion pressures during CPR in swine. Resuscitation 1993;26:3—12.
258. Buunk G, van der Hoeven JG, Meinders AE. Cerebrovascular reactivity in comatose patients resuscitated from a cardiac arrest. Stroke 1997;28:1569—73.
259. Aufderheide TP, Sigurdsson G, Pirrallo RG, et al. Hyperventilation-induced hypotension during cardiopulmonary resuscitation. Circulation 2004;109:1960—5.
260. Muizelaar JP, Marmarou A, Ward JD, et al. Adverse effects of prolonged hyperventilation in patients with severe head injury: a randomized clinical trial. J Neurosurg 1991;75:731—9.
261. Carmona Suazo JA, Maas AI, van den Brink WA, van Santbrink H, Steyerberg EW, Avezaat CJ. CO_2 reactivity and brain oxygen pressure monitoring in severe head injury. Crit Care Med 2000;28:3268—74.
262. Schneider GH, Sarrafzadeh AS, Kiening KL, Bardt TF, Unterberg AW, Lanksch WR. Influence of hyperventilation on brain tissue-PO2, PCO2, and pH in patients with intracranial hypertension. Acta Neurochir Suppl 1998;71:62—5.
263. Unterberg AW, Kiening KL, Hartl R, Bardt T, Sarrafzadeh AS, Lanksch WR. Multimodal monitoring in patients with head injury: evaluation of the effects of treatment on cerebral oxygenation. J Trauma 1997;42:S32—7.
264. Skippen P, Seear M, Poskitt K, et al. Effect of hyperventilation on regional cerebral blood flow in head-injured children. Crit Care Med 1997;25:1402—9.
265. Hutchinson PJ, Gupta AK, Fryer TF, et al. Correlation between cerebral blood flow, substrate delivery, and metabolism in head injury: a combined microdialysis and triple oxygen positron emission tomography study. J Cereb Blood Flow Metab 2002;22:735—45.
266. Coles JP, Minhas PS, Fryer TD, et al. Effect of hyperventilation on cerebral blood flow in traumatic head injury: clinical relevance and monitoring correlates. Crit Care Med 2002;30:1950—9.
267. Dings J, Meixensberger J, Amschler J, Roosen K. Continuous monitoring of brain tissue PO2: a new tool to minimize the risk of ischemia caused by hyperventilation therapy. Zentralbl Neurochir 1996;57:177—83.
268. Cold GE. Measurements of CO_2 reactivity and barbiturate reactivity in patients with severe head injury. Acta Neurochir (Wien) 1989;98:153—63.
269. Zhang S, Zhi D, Lin X, Shang Y, Niu Y. Effect of mild hypothermia on partial pressure of oxygen in brain tissue and brain temperature in patients with severe head injury. Chin J Traumatol 2002;5:43—5.
270. Obrist WD, Langfitt TW, Jaggi JL, Cruz J, Gennarelli TA. Cerebral blood flow and metabolism in comatose patients with acute head injury. Relationship to intracranial hypertension. J Neurosurg 1984;61:241—53.
271. Hickey RW, Kochanek PM, Ferimer H, Graham SH, Safar P. Hypothermia and hyperthermia in children after resuscitation from cardiac arrest. Pediatrics 2000;106(Pt 1):118—22.
272. Hypothermia After Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. N Engl J Med 2002;346:549—56.
273. Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. N Engl J Med 2002;346:557—63.
274. Gluckman PD, Wyatt JS, Azzopardi D, et al. Selective head cooling with mild systemic hypothermia after neonatal encephalopathy: multicentre randomised trial. Lancet 2005;365:663—70.
275. Battin MR, Penrice J, Gunn TR, Gunn AJ. Treatment of term infants with head cooling and mild systemic hypothermia (35.0 degrees C and 34.5 degrees C) after perinatal asphyxia. Pediatrics 2003;111:244—51.
276. Compagnoni G, Pogliani L, Lista G, Castoldi F, Fontana P, Mosca F. Hypothermia reduces neurological damage in asphyxiated newborn infants. Biol Neonate 2002;82:222—7.
277. Debillon T, Daoud P, Durand P, et al. Whole-body cooling after perinatal asphyxia: a pilot study in term neonates. Dev Med Child Neurol 2003;45:17—23.
278. Gunn AJ, Gluckman PD, Gunn TR. Selective head cooling in newborn infants after perinatal asphyxia: a safety study. Pediatrics 1998;102:885—92.
279. Albrecht IInd RF, Wass CT, Lanier WL. Occurrence of potentially detrimental temperature alterations in hospitalized patients at risk for brain injury. Mayo Clin Proc 1998;73:629—35.
280. Takino M, Okada Y. Hyperthermia following cardiopulmonary resuscitation. Intensive Care Med 1991;17:419—20.
281. Takasu A, Saitoh D, Kaneko N, Sakamoto T, Okada Y. Hyperthermia: is it an ominous sign after cardiac arrest? Resuscitation 2001;49:273—7.
282. Zeiner A, Holzer M, Sterz F, et al. Hyperthermia after cardiac arrest is associated with an unfavorable neurologic outcome. Arch Intern Med 2001;161:2007—12.

283. Hickey RW, Kochanek PM, Ferimer H, Alexander HL, Garman RH, Graham SH. Induced hyperthermia exacerbates neurologic neuronal histologic damage after asphyxial cardiac arrest in rats. Crit Care Med 2003;31:531—5.
284. Coimbra C, Boris-Moller F, Drake M, Wieloch T. Diminished neuronal damage in the rat brain by late treatment with the antipyretic drug dipyrone or cooling following cerebral ischemia. Acta Neuropathol (Berl) 1996;92:447—53.
285. Coimbra C, Drake M, Boris-Moller F, Wieloch T. Long-lasting neuroprotective effect of postischemic hypothermia and treatment with an anti-inflammatory/antipyretic drug: evidence for chronic encephalopathic processes following ischemia. Stroke 1996;27:1578—85.
286. Hildebrand CA, Hartmann AG, Arcinue EL, Gomez RJ, Bing RJ. Cardiac performance in pediatric near-drowning. Crit Care Med 1988;16:331—5.
287. Checchia PA, Sehra R, Moynihan J, Daher N, Tang W, Weil MH. Myocardial injury in children following resuscitation after cardiac arrest. Resuscitation 2003;57:131—7.
288. Rivers EP, Wortsman J, Rady MY, Blake HC, McGeorge FT, Buderer NM. The effect of the total cumulative epinephrine dose administered during human CPR on hemodynamic, oxygen transport, and utilization variables in the postresuscitation period. Chest 1994;106:1499—507.
289. Mullner M, Domanovits H, Sterz F, et al. Measurement of myocardial contractility following successful resuscitation: quantitated left ventricular systolic function utilising non-invasive wall stress analysis. Resuscitation 1998;39:51—9.
290. Laurent I, Monchi M, Chiche JD, et al. Reversible myocardial dysfunction in survivors of out-of-hospital cardiac arrest. J Am Coll Cardiol 2002;40:2110—6.
291. Kamohara T, Weil MH, Tang W, et al. A comparison of myocardial function after primary cardiac and primary asphyxial cardiac arrest. Am J Respir Crit Care Med 2001;164:1221—4.
292. Gazmuri RJ, Weil MH, Bisera J, Tang W, Fukui M, McKee D. Myocardial dysfunction after successful resuscitation from cardiac arrest. Crit Care Med 1996;24:992—1000.
293. Lobato EB, Gravenstein N, Martin TD. Milrinone, not epinephrine, improves left ventricular compliance after cardiopulmonary bypass. J Cardiothorac Vasc Anesth 2000;14:374—7.
294. Kern KB, Hilwig RW, Berg RA, et al. Postresuscitation left ventricular systolic and diastolic dysfunction: treatment with dobutamine. Circulation 1997;95:2610—3.
295. Meyer RJ, Kern KB, Berg RA, Hilwig RW, Ewy GA. Post-resuscitation right ventricular dysfunction: delineation and treatment with dobutamine. Resuscitation 2002;55:187—91.
296. Niemann JT, Garner D, Khaleeli E, Lewis RJ. Milrinone facilitates resuscitation from cardiac arrest and attenuates postresuscitation myocardial dysfunction. Circulation 2003;108:3031—5.
297. Hoffman TM, Wernovsky G, Atz AM, et al. Efficacy and safety of milrinone in preventing low cardiac output syndrome in infants and children after corrective surgery for congenital heart disease. Circulation 2003;107:996—1002.
298. Innes PA, Frazer RS, Booker PD, et al. Comparison of the haemodynamic effects of dobutamine with enoximone after open heart surgery in small children. Br J Anaesth 1994;72:77—81.
299. Laitinen P, Happonen JM, Sairanen H, Peltola K, Rautiainen P. Amrinone versus dopamine and nitroglycerin in neonates after arterial switch operation for transposition of the great arteries. J Cardiothorac Vasc Anesth 1999;13:186—90.
300. Abdallah I, Shawky H. A randomised controlled trial comparing milrinone and epinephrine as inotropes in paediatric patients undergoing total correction of Tetralogy of Fallot. Egypt J Anaesth 2003;19(4):323—9.
301. Butterworth IV JF, Royster RL, Prielipp RC, Lawless ST, Wallenhaupt SL. Amrinone in cardiac surgical patients with left-ventricular dysfunction. A prospective, randomized placebo-controlled trial. Chest 1993;104:1660—7.
302. Kikura M, Sato S. The efficacy of preemptive Milrinone or Amrinone therapy in patients undergoing coronary artery bypass grafting. Anesth Analg 2002;94:22—30 [table of contents].
303. Calle PA, Buylaert WA, Vanhaute OA. Glycemia in the post-resuscitation period. The Cerebral Resuscitation Study Group. Resuscitation 1989;17(Suppl.):S181—8 [discussion S99—206].
304. Langhelle A, Tyvold SS, Lexow K, Hapnes SA, Sunde K, Steen PA. In-hospital factors associated with improved outcome after out-of-hospital cardiac arrest. A comparison between four regions in Norway. Resuscitation 2003;56:247—63.
305. Longstreth Jr WT, Diehr P, Inui TS. Prediction of awakening after out-of-hospital cardiac arrest. N Engl J Med 1983;308:1378—82.
306. Longstreth Jr WT, Inui TS. High blood glucose level on hospital admission and poor neurological recovery after cardiac arrest. Ann Neurol 1984;15:59—63.
307. Mullner M, Sterz F, Binder M, Schreiber W, Deimel A, Laggner AN. Blood glucose concentration after cardiopulmonary resuscitation influences functional neurological recovery in human cardiac arrest survivors. J Cereb Blood Flow Metab 1997;17:430—6.
308. Skrifvars MB, Pettila V, Rosenberg PH, Castren M. A multiple logistic regression analysis of in-hospital factors related to survival at six months in patients resuscitated from out-of-hospital ventricular fibrillation. Resuscitation 2003;59:319—28.
309. Losek JD. Hypoglycemia and the ABC'S (sugar) of pediatric resuscitation. Ann Emerg Med 2000;35:43—6.
310. Srinivasan V, Spinella PC, Drott HR, Roth CL, Helfaer MA, Nadkarni V. Association of timing, duration, and intensity of hyperglycemia with intensive care unit mortality in critically ill children. Pediatr Crit Care Med 2004;5:329—36.
311. van den Berghe G, Wouters P, Weekers F, et al. Intensive insulin therapy in the critically ill patients. N Engl J Med 2001;345:1359—67.
312. Hoxworth JM, Xu K, Zhou Y, Lust WD, LaManna JC. Cerebral metabolic profile, selective neuron loss, and survival of acute and chronic hyperglycemic rats following cardiac arrest and resuscitation. Brain Res 1999;821:467—79.
313. D'Alecy LG, Lundy EF, Barton KJ, Zelenock GB. Dextrose containing intravenous fluid impairs outcome and increases death after eight minutes of cardiac arrest and resuscitation in dogs. Surgery 1986;100:505—11.
314. Nakakimura K, Fleischer JE, Drummond JC, et al. Glucose administration before cardiac arrest worsens neurologic outcome in cats. Anesthesiology 1990;72:1005—11.
315. Farias LA, Willis M, Gregory GA. Effects of fructose-1,6-diphosphate, glucose, and saline on cardiac resuscitation. Anesthesiology 1986;65:595—601.
316. Natale JE, Stante SM, D'Alecy LG. Elevated brain lactate accumulation and increased neurologic deficit are associated with modest hyperglycemia in global brain ischemia. Resuscitation 1990;19:271—89.
317. Longstreth Jr WT, Copass MK, Dennis LK, Rauch-Matthews ME, Stark MS, Cobb LA. Intravenous glucose after out-of-

hospital cardiopulmonary arrest: a community-based randomized trial. Neurology 1993;43:2534—41.
318. Vannucci RC, Vannucci SJ. Hypoglycemic brain injury. Semin Neonatol 2001;6:147—55.
319. Salhab WA, Wyckoff MH, Laptook AR, Perlman JM. Initial hypoglycemia and neonatal brain injury in term infants with severe fetal acidemia. Pediatrics 2004;114:361—6.
320. Lin Y, Greisen G. Analysis of the risk of brain damage in asphyxiated infants. J Perinat Med 1996;24:581—9.
321. Mir NA, Faquih AM, Legnain M. Perinatal risk factors in birth asphyxia: relationship of obstetric and neonatal complications to neonatal mortality in 16,365 consecutive live births. Asia Oceania J Obstet Gynaecol 1989;15:351—7.
322. Ondoa-Onama C, Tumwine JK. Immediate outcome of babies with low Apgar score in Mulago Hospital, Uganda. East Afr Med J 2003;80:22—9.
323. Longstreth Jr WT, Diehr P, Cobb LA, Hanson RW, Blair AD. Neurologic outcome and blood glucose levels during out-of-hospital cardiopulmonary resuscitation. Neurology 1986;36:1186—91.
324. Gillis J, Dickson D, Rieder M, Steward D, Edmonds J. Results of inpatient pediatric resuscitation. Crit Care Med 1986;14:469—71.
325. Schindler MB, Bohn D, Cox PN, et al. Outcome of out-of-hospital cardiac or respiratory arrest in children. N Engl J Med 1996;335:1473—9.
326. Suominen P, Korpela R, Kuisma M, Silfvast T, Olkkola KT. Paediatric cardiac arrest and resuscitation provided by physician-staffed emergency care units. Acta Anaesthesiol Scand 1997;41:260—5.
327. Suominen P, Olkkola KT, Voipio V, Korpela R, Palo R, Rasanen J. Utstein style reporting of in-hospital paediatric cardiopulmonary resuscitation. Resuscitation 2000;45:17—25.
328. Lopez-Herce J, Garcia C, Dominguez P, et al. Characteristics and outcome of cardiorespiratory arrest in children. Resuscitation 2004;63:311—20.
329. Reis AG, Nadkarni V, Perondi MB, Grisi S, Berg RA. A prospective investigation into the epidemiology of in-hospital pediatric cardiopulmonary resuscitation using the international Utstein reporting style. Pediatrics 2002; 109:200—9.
330. Idris AH, Berg RA, Bierens J, et al. Recommended guidelines for uniform reporting of data from drowning: The ''Utstein style''. Resuscitation 2003;59:45—57.
331. Morris MC, Wernovsky G, Nadkarni VM. Survival outcomes after extracorporeal cardiopulmonary resuscitation instituted during active chest compressions following refractory in-hospital pediatric cardiac arrest. Pediatr Crit Care Med 2004;5:440—6.
332. Dalton HJ, Siewers RD, Fuhrman BP, et al. Extracorporeal membrane oxygenation for cardiac rescue in children with severe myocardial dysfunction. Crit Care Med 1993;21:1020—8.
333. del Nido PJ. Extracorporeal membrane oxygenation for cardiac support in children. Ann Thorac Surg 1996;61:336—9 [discussion 40—1].
334. Duncan BW, Ibrahim AE, Hraska V, et al. Use of rapid-deployment extracorporeal membrane oxygenation for the resuscitation of pediatric patients with heart disease after cardiac arrest. J Thorac Cardiovasc Surg 1998;116:305—11.
335. Parra DA, Totapally BR, Zahn E, et al. Outcome of cardiopulmonary resuscitation in a pediatric cardiac intensive care unit. Crit Care Med 2000;28:3296—300.
336. Aharon AS, Drinkwater Jr DC, Churchwell KB, et al. Extracorporeal membrane oxygenation in children after repair of congenital cardiac lesions. Ann Thorac Surg 2001;72:2095—101 [discussion 101—2].
337. Hazinski MF, Chahine AA, Holcomb IIIrd GW, Morris Jr JA. Outcome of cardiovascular collapse in pediatric blunt trauma. Ann Emerg Med 1994;23:1229—35.